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THE CHANGING MOLE

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THE threshold of suspicion for malignant disease is probably lower now among American physicians and laymen than at any previous time. There is more study and discussion of malignant processes. Much of this zeal springs from the interest and activity of organizations such as the American Cancer Society, which is devoting a great deal of energy in obtaining financial support, to be allocated to worthwhile projects in research institutions. The federally supported National Cancer Institute has for years carried out research in the field of malignancy. Many universities and hospitals have maintained cancer clinics, at which the skill and knowledge of physicians in various fields of medicine are pooled in the interest of the patients with possible malignant disease. There are many smaller organizations dedicated to research and treatment of cancer.

There is most interest in real cancer (carcinoma), including cancer of the skin, because of its frequency, but malignant neoplasia of other tissues has by no means been neglected. We are interested today in a neoplasm which is less frequently seen, namely, melanoma, and, specifically, melanoma originating in the skin. Its infrequency is both fortunate and unfortunate; fortunate for our patients, but unfortunate for physicians, because of limited opportunity for becoming familiar with it, so that, at present, the threshold of suspicion is maintained at too high a level. Miescher found that, at the University of Zurich, Switzerland, cutaneous carcinoma outnumbered melanoma 20:1. In areas with a high proportion of sunshiny days and a correspondingly greater incidence of cutaneous carcinomas, the ratio would undoubtedly be much higher.

For our purpose today, the word "mole" is used in the sense of a pigmented nevus,[5] along with its non-pigmented relative, the non-pigmented nevus. By "changing mole" we mean a nevus exhibiting clinical and/or microscopic alteration leading to malignant melanoma, commonly known simply as "melanoma." A pigmented nevus is best considered as a benign tumor of pigment-forming cells, which are known as melanoblasts. Melanoma is a malignant tumor of pigment-forming cells. These cells originate from the neural crest during embryonic life, from which location they wander out to the skin, the eyes and the pia mater about the medulla. Melanoma which has originated in other parts of the body must have arisen from misplaced melanoblasts, or may constitute a misnamed tumor, such as "melanoma" of the adrenal gland, which most authorities believe is not a true melanoma. Melanoma resembles nervous tumors of a malignant nature in its resistance to roentgen and radium irradiation, in contrast to cancer of the skin, which is relatively radiosensitive.

Pigmented Nevi

Pigmented nevi are among the most common of benign tumors. Pack[14] found that each adult person has an average of twenty such lesions. They are located more frequently on the face, neck, trunk and upper extremities, infrequently on the genitalia and feet, and are almost unknown on mucous surfaces. Melanoma, on the other hand, is most frequent on the face, genitalia and feet,

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and does occur, but less often, on mucous surfaces. This contrast between the sites of predilection for nevi and melanoma makes one wonder whether such malignancy does originate most frequently in nevi, as has been stated by so many authors.

Pigmented nevi vary from smooth brown macules through elevated papular tumors to large hairy lesions covering much of the body (bathing trunk nevus). The mole which becomes malignant is usually the smooth brown lesion. Elevated nevi, with or without hairs are not common offenders. However, extensive moles have become malignant, even before puberty. They also are often associated with melanosis of the pia mater about the medulla, which may result in benign or malignant tumor of the area, with symptoms of brain tumor.

Microscopically, according to Lund and Stobbe[12] a young nevus consists of nevus cells, containing a varying amount of melanin, in close apposition to the epidermis, with or without unpigmented cells deeper in the dermis. In the older nevi, all cells may be deep in the skin. This variety of nevus is the one most frequently seen, and is the common mole of Traub and Keil.[21] The nevus cells may be fusiform or oval, and resemble melanoblasts rather than epidermal cells. Nevi in which there is active multiplication of nevus cells at the epidermo-dermal junction are known as active nevi. The designation "junction type nevus," frequently seen nowadays, includes quiescent nevi with nevus cells at the epidermo-dermal junction, active nevi with cells in the same location, and lentigo maligna; hence, both benign and malignant growths. Therefore it cannot be an accepted term. Some non-pigmented nevi resemble neurofibromas.

Treatment of Nevi

The consensus is that moles at sites of irritation should be removed, preferably before puberty. Surgical excision is satisfactory, and is always indicated for nevi that are enlarging or becoming darker. My own preference[4] is for snipping off the superficial portion of an elevated quiescent nevus and cauterizing the base for a good cosmetic result. The removed portion is always biopsied, so that any malignant change can be determined. All excised tumors should be sent to the laboratory for biopsy.

Acquired Nevi

Acquired nevi are those which appear after early childhood. A good illustration is the remarkable case of Ebert,[9] as follows:

Case 1.—A boy, aged twelve, presented several hundred papules, first noted eight months previously on the covered parts of the body and the backs of the hands, with a predilection for the extensor surfaces of the arms, the buttocks and the posterior surfaces of the thighs. They were increasing in number and varied in color from flesh color to brown. Over a period of two years one of the lesions on the buttock became coal black and many others became black and assumed the appearance of ordinary pigmented nevi. Four years after first being seen, no new lesions were appearing. Some of the original ones were brownish red and some were coal black.

Microscopic examination showed the tumors to be composed of cells having the appearance of young nevus cells, some of which were still in close association with the epidermis and others were in masses in the dermis. They were all strongly dopa positive, although very little melanin had formed as yet.

Mongolian Spot and Blue Nevus

Mongolian spots (sacral spots) and blue nevi are bluish macular or slightly elevated lesions containing pigmented melanoblasts in the dermis. Mongolian spots are present at birth in a large percentage of children of the dark races and tend to fade out over a few years. Blue nevi are smaller and darker and may appear at any time during early life. I have never seen melanoma develop in a blue nevus, and the reported cases of such development are not very convincing. Treatment of blue nevus is excision.

Precipitating Factors in Melanoma

The cause of melanoma is that of malignant disease in general, many etiologic factors of which are only imperfectly understood. Family history is almost unknown. The unusual reports of family incidence suggest that occurrence in more than one member is probably coincidental. Sunlight does not seem to be prominent as a precipitating factor, although the common appearance on the face has been advanced in its support. Irritation appears to play a definite role. Daland and Holmes[8] reported the influence of trauma in seventy of 174 cases, Amadon[2] in eleven of twenty-seven cases. Herold[11] reported the prominent role of trauma in African Negroes, in whom most melanomas originate on the soles,

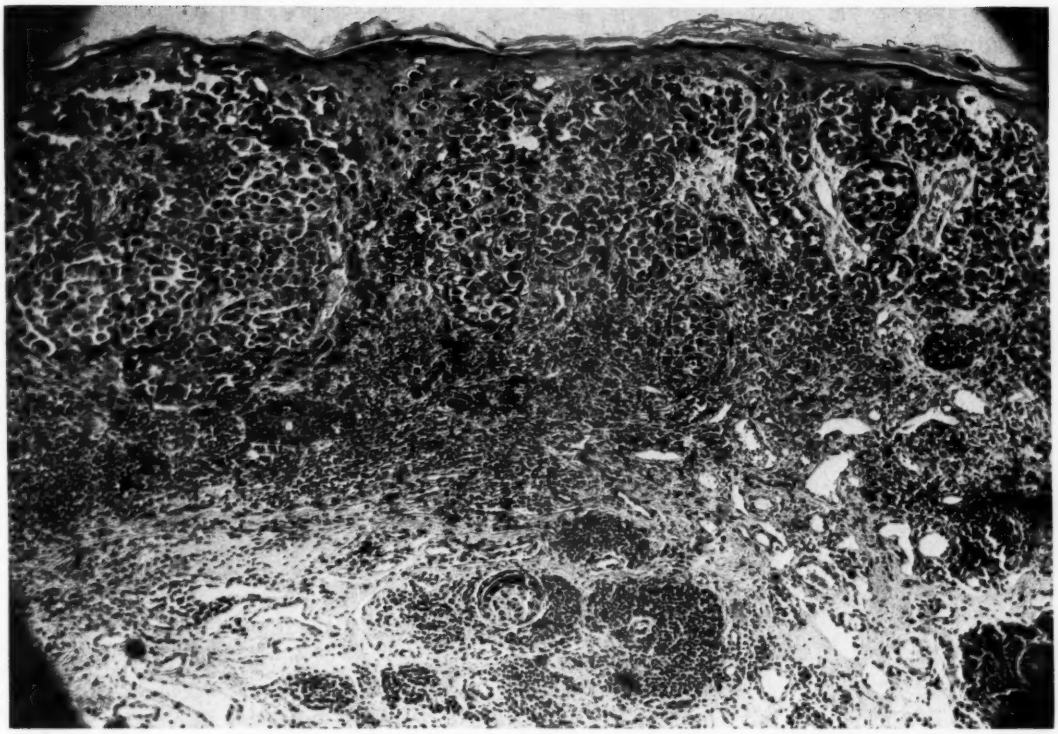

Fig. 1. Melanoma arising in nevus. Melanoma cells and lymphocytes are superficial and nevus cells are deep. (H&E stain, high power).

following injury from sharp objects, such as thorns.

According to Scharnagel and Pack,[18] puberty and pregnancy are periods of altered hormone balance which stimulates the formation and spread of melanoma. Spitz[20] called attention to the relative benignity of melanomas before puberty, and for this reason separated them from ordinary melanomas under the designation "juvenile melanomas." The possible stimulating effect of puberty and the probable stimulation during pregnancy are illustrated in the following case report (Ref. 4, Case 2):

Case 2.—A girl, aged sixteen, had always had a small pigmented nevus on her right shoulder. It had occasionally become tender because of irritation by a shoulder strap, had bled on occasions, and had been slowly enlarging. She presented a slightly elevated papule, brown in color, 3.0 by 5.0 mm. in size. The lesion was completely removed locally. Microscopic examination showed a superficial nevus, the center of which was composed of a solid mass of cells which was encroaching on both the epidermis and dermis. Diagnosis was made of early melanoma and the area was excised surgically. Seven years later, during pregnancy, a tumor was noted on the opposite side of the thorax in the region of a lower rib. It was removed surgically and was found to be a partially pigmented melanoma. In order to avoid further stimulating effect of pregnancy, cesarean section was performed at the eighth month and a normal baby was delivered. During a subsequent period of over one year, no further signs of melanoma have been observed. However, to avoid stimulation of any possible remaining melanoma cells, she has been advised to avoid further pregnancy.

Melanoma from Nevus

Determination of the actual percentage of melanomas that originate in pigmented nevi is difficult. If accurate history reveals that a pigmented lesion has been present since birth or early childhood, it must be assumed that a nevus existed. If both nevus cells and melanoma cells are found on section (Fig. 1), it is certain that a nevus has been present. Microscopic sections are not always adequate, because diagnosis is usually made on only a few sections, which may not show nevus cells if the melanoma has outgrown the nevus. Serial sections would be more informative.

Careful study has shown that melanoma can be proven to have originated in a pre-existing nevus in not over 25 per cent of instances. And even so, the actual malignant cells have their origin in normal melanoblasts at the epidermodermal junction rather than in nevus cells themselves. Pathologic theory states that malignant cells do not arise directly from benign neoplastic cells. Daland and Holmes[8] found that only 18 per cent of melanomas originated in pigmented nevi. In specimens examined in the Dermatological Laboratory of the University of Chicago, about 23 per cent can be seen to originate from nevi. Miescher, at the University of Zurich, Switzerland, found such origin in 25 per cent of cases.

Lentigo Maligna

Clinically, melanoma originates in the other 75 per cent of cases as a brown macule, at first smooth and non-elevated, which was originally described by Johnathan Hutchinson as a "malignant freckle." Dubreuilh translated this designation into French as "lentigo malign," which in English is "lentigo maligna." "Lentigo" means an acquired brown spot, and "maligna" denotes its malignant nature. Lentigo maligna appears after puberty, enlarges slowly and unevenly, and shows various degrees of light and dark brown color. After a few months or years, serum exudes and, eventually, bleeding occurs. Later, a tumor forms, which may be pigmented or non-pigmented melanoma. The following case report is illustrative:

Case 3.—A housewife, aged forty-nine, had developed a small dark brown spot on the lateral surface of the right ankle about four or five years previously. It enlarged irregularly, and she had been referred by her family physician to a dermatologist one and one half years before being seen. He had advised observation. Three or four months ago serum oozed from the lesion and it later bled. She consulted a second dermatologist, who removed a specimen for biopsy. Microscopic examination (Fig. 2) showed pronounced round cell infiltrate in the superficial dermis. At the epidermodermal junction were large numbers of large, succulent, partly pigmented cells, which were working their way to the epidermal surface and also penetrating into the dermis. This is typical for lentigo maligna. When seen, she presented a geographic lesion 2.0 by 3.0 cm. in size, very slightly elevated, with a color varying in different parts from light to dark brown. The area was widely removed surgically and the defect was skin-grafted.

Of course, it must be remembered that pigmented nevi also enlarge as the person becomes older, become slightly darker and, if injured sufficiently, will bleed. A nevus in which melanoma has developed enlarges more rapidly, becomes more deeply brown or black, and tends to bleed on slighter trauma. Pigmented melanoma can only be diagnosed clinically in 50 per cent of cases. Biopsy is essential.

Non-pigmented Melanoma

Non-pigmented melanoma appears in a variety of forms. Some of them simulate verruca vulgaris, especially one that has been irritated or infected. The most common picture is that of a reddish, soft tumor which bleeds on trauma, and closely simulates granuloma pyogenicum. The following case report is illustrative:

Case 4.—A woman, aged forty-three, a farmer's wife, had had a mole on her calf all her life. It had grown in the past two years and had bled after having been pecked by a chicken on two occasions. Clinical diagnosis was made of granuloma pyogenicum. The tumor was excised. Microscopic examination showed non-pigmented melanoma. Diagnosis of non-pigmented melanoma can only be made by biopsy.

How has the misconception arisen that most melanomas have originated in pigmented nevi. Several reasons seem apparent:

1. Lack of careful history. With the exception of the acquired variety, which is rare, nevi appear early in life.
2. Lack of careful clinical examination. Quiescent nevi can usually be differentiated from the more rapidly growing lentigo maligna or melanoma.
3. Failure to biopsy all tumors. This procedure is now considered a *sine qua non* for accurate diagnosis of melanoma. Biopsy is safe if adequate treatment is carried out as soon as microscopic diagnosis is made.

Clinical Course of Melanoma

The clinical course of melanoma is inclined to be regular. There is first of all relatively rapid local growth. There is at times local lymphatic extension appearing in the form of satellite lesions, ordinarily along the lymphatics draining the area. The regional lymph nodes will eventually become enlarged. Only biopsy will reveal the presence of melanoma cells in such nodes.

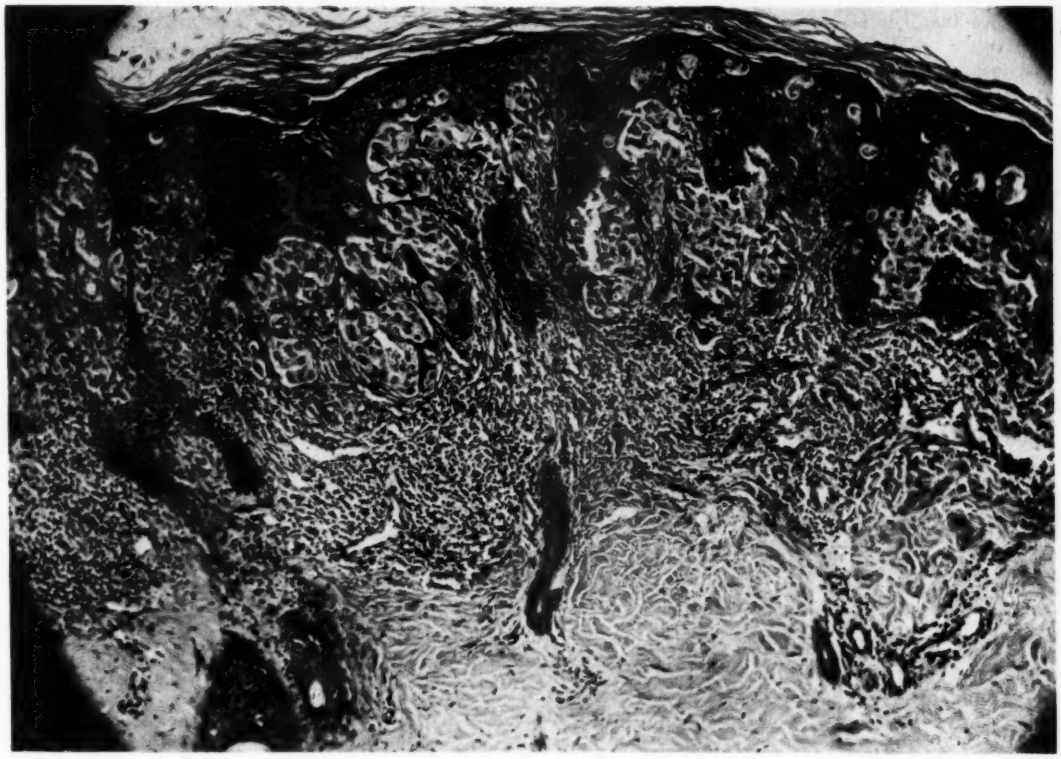

Fig. 2. Lentigo maligna. Melanoma cells and lymphocytes are superficial. No nevus cells are present. (H&E stain, high power).

Systemic dissemination will subsequently follow, with tumor cells appearing in practically any tissue of the body. The urine may become darkened (melanuria), and the entire body may become practically black (systemic melanosis). Ackerman[1] stated that 10 per cent of melanomas are first noted as metastatic growths in the regional lymph nodes. Further search ordinarily reveals a primary lesion or a history is obtained that a "mole" had been removed in the area of drainage. Selig[19] reported removal of a metastatic non-pigmented melanomatous mass from the groin of a forty-seven-year-old woman. Careful search of the lower limb revealed no primary growth. Four and one-quarter years later, a painless ulcer appeared on the right heel, which also showed non-pigmented melanoma on biopsy.

Mucosal Melanoma

Melanoma may appear on any of the mucocutaneous junctions or adjacent mucous surfaces. Since pigmented nevi are almost never seen on mucosal surfaces, the origin is from a blackish macule corresponding to the cutaneous lentigo maligna. Spread to regional lymph nodes seems to appear somewhat sooner than from the cutaneous surface, possibly because of greater vascularity of the regions.

Treatment of Melanoma

Treatment of melanoma consists of removal. Various other therapeutic measures, such as roentgen and radium irradiation, administration of Coley's fluid, colloidal lead, and various radioactive isotopes have been unsatisfactory. Soon after Willy Meyer and Halsted[10] independently, in 1894, introduced the principle of dissection in continuity, i.e., removal of the breast, lymphatic channels and lymph nodes in one piece, the method was applied by Pringle[16] to treatment of melanoma:

> A girl, aged seventeen, presented a melanoma on the left forearm of two years' duration. There had been no pre-existing mole. In 1898, a strip of tissue 2 inches in width was excised and the axillary lymph nodes were removed. They were found to contain metastatic tumor growth. She was again presented[17] thirty-eight years

after operation, during which time she had had no recurrence. A second patient, a man, aged thirty, presented a melanoma, 3.0 by 4.0 cm. in size, of two years' duration, on the left thigh, originating in a mole that had been present since birth. The primary growth was removed, along with a strip of tissue up to and including the lymph nodes as far as the bifurcation of the common iliac artery. Metastatic melanoma was found in the lower iliac but not in the upper iliac nodes. The patient was redemonstrated thirty years later,[17] during which time he had had no recurrence.

The same principle of dissection in continuity was applied to malignancy of the rectum by Sir Ernest Miles[13] and to the vulva by Bassett.[3] It is useful for melanoma occurring in these regions. Pack, Scharnagel and Morfit[15] stated that application of this method to treatment of melanoma requires considerable ingenuity, because the malignant melanomas are so variable in their location and drain into so many groups of regional lymph nodes. Pack[14] has recommended an even more radical operation in some instances of advanced melanoma of the extremities, namely, disarticulation at the hip or amputation of the arm and shoulder girdle, as the case may be. Many surgeons hesitate to perform such a radical operation, even though they would not hesitate to amputate for sarcoma of the bone.

Prognosis of Melanoma

Prognosis of melanoma was formerly practically hopeless. Boulay[6] in 1888 collected 191 cases from the literature, of which 152 had been operated on. From the clinical descriptions, the process was usually already far advanced and hopeless, but, even so, there were a few survivors following radical surgical operation. Following radical surgical treatment, Pack,[14] in 1948, stated that in the past fifteen years, their percentage of patients who have survived five years without recurrence, that is to say, the incidence of so-called definitive cures, had increased 600 per cent, surely an improvement far greater than that obtained with almost any other common variety of cancer. Still, Pack's percentage of five-year survivals without metastases to lymph nodes is only 18 per cent, and for melanoma metastatic to regional lymph nodes and treated by surgical means is only 15 per cent. Five-year survival is not as significant in melanoma as in some other malignant tumors because of the tendency to late recurrence. Wilbur and Hartman[22] found local recurrence as late as nine years and nodal metastasis as late as thirteen years. However, with early diagnosis and radical surgical treatment, the prognosis should improve greatly. Many lives can be saved by prompt and adequate diagnosis and treatment. Capra and Ewing[7] recently stated: "Serious as is the outlook for the patient with a malignant melanoma, heroic resection will often salvage what appears to be the most hopeless case."

Summary

1. Melanoma arises in a pigmented mole in not over 25 per cent of cases; it usually originates in normal skin in the form of lentigo maligna.

2. All suspected lesions must be biopsied.

3. Post-pubertal melanoma is more serious than the prepubertal variety.

4. Treatment of melanoma is surgical, and must be radical.

5. Prognosis is greatly improved by earlier diagnosis and more radical surgical procedures.

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CONGENITAL OBSTRUCTION OF THE ALIMENTARY CANAL IN THE NEWBORN

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CONGENTIAL obstruction of the alimentary canal has received increasing consideration during the past few years since it has been demonstrated that the various types of such obstruction are amenable to surgical treatment. It is now evident that these lesions are much more common than previous reports would indicate, and in all probability many congenital obstructions of the alimentary canal are still being overlooked. Evans[1] in an exhaustive study of atresia of the gastrointestinal tract, including the esophagus and ano-rectal region, estimates that this particular anomaly occurs in approximately one in every 1,500 births. He believes "it is less considered and more unheeded, than uncommon."

The authors wish to report the incidence and cases of congenital obstruction of the alimentary canal among the 4,374 live births in the St. Olaf Hospital, Austin, Minnesota, during the five-year-period ending December 31, 1950. It is our opinion that these congenital anomalies may be more easily overlooked in a predominantly rural area, such as ours, where a special academic interest in such matters may be lacking. It is particularly important that general physicians, who supervise the care of the majority of newborn infants, be especially alerted to the problem.

A review of the 4,374 live-births in the St. Olaf Hospital for the five-year period ending December 31, 1950, was undertaken by one of us (R.R.W.). There were seventy-four neonatal deaths, an incidence of 16.91 per 1,000 live births. This compares very favorably with the reported incidence of neonatal deaths per live births from other surveys. A careful review was made of each neonatal death. Only one of these neonatal deaths could be attributed to congenital obstruction of the alimentary canal. This case is reported below.

There were three newborn infants with complete congenital obstruction of the alimentary canal who survived. A brief report of each patient is given below. Fourteen infants with congenital pyloric stenosis, all treated successfully by operation, were seen by the authors during the five-year period of this survey. These cases are not specifically included in this report, nor does the number of cases of this disease necessarily represent its true incidence in relation to the number of live births.

Case Reports

Case 1.—R. D. Birth weight, 7 pounds. Girl.

The diagnosis of complete atresia of the third portion of the duodenum was established by the family physician, Dr. C. L. Sheedy. Duodenojejunostomy was performed when the infant was fifty-eight hours old. Recovery was uneventful. The infant was discharged on her eighth postoperative day. This patient is now a healthy, normal child, three and one half years of age.

Case 2.—F. F. Birth weight, 5 pounds 10½ ounces. Boy.

The diagnosis of congenital atresia of the esophagus with a tracheoesophageal fistula was made within forty-eight hours of birth by Dr. F. H. Rosenthal. The infant had episodes of cyanosis and choking due to excessive thick mucus in the pharynx. An end-to-end esophageal anastomosis with closure of the tracheo-esophageal fistula, without an associated gastrostomy, was performed four days after birth. The infant was discharged on the eleventh postoperative day. Several esophageal dilatations with a Philip's urethral bougie were performed as office procedures. An ano-rectal stenosis also required several dilatations. This patient is now a healthy child, ten months of age.

Case 3.—B. N. Birth weight, 6 pounds, 13 ounces. Girl.

Symptoms of partial intestinal obstruction dated from birth, but the diagnosis of complete obstruction was not made until the eighth day. Operation revealed a duodenal obstruction, partial, due to incomplete rotation of the cecum and an associated volvulus of the midgut. The operation described by Ladd and Gross[2] was performed. The infant was discharged on the tenth postoperative day. She is now a healthy infant, seven months of age.

Case 4.—B. H. Birth weight, 5 pounds 10 ounces. Girl.

The diagnosis of congenital atresia of the esophagus with tracheoesophageal fistula was not established until forty-eight hours after birth. The infant had excessive mucus, cyanosis and difficulty in breathing the day of birth and died three days after birth. Operation was not performed because of the patient's poor condition. An autopsy was not obtained.

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THE USE OF ADRENOCORTICOTROPHIC HORMONE IN THE TREATMENT OF MYASTHENIA GRAVIS

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"I have now a prudent and honest woman in cure, who for many years has been obnoxious to this kind of bastard palsy, not only in her limbs but likewise in her tongue. This person for some time speaks freely and readily enough, but after long, hasty or laborious speaking presently she becomes mute as a fish, and cannot bring forth a word. Nay, and does not recover the use of her voice until after many minutes."

THUS, in 1685, Willis[1] in somewhat graphic terminology recorded the first description of a patient with myasthenia gravis. Basic contributions to the knowledge of this condition have subsequently been made, but not until 1934 was there any significant advance in the treatment of this "bastard palsy." Walker[9,10] in that year made her classical report on the use of eserine and then subsequently prostigmine in the treatment of the symptoms of myasthenia gravis. Other therapies have been used which give additional aid to some patients with myasthenia gravis; none of these, however, has supplanted prostigmine. With the advent of ACTH it was hoped that a new therapeutic era might be opened to patients with myasthenia gravis.

Although less than thirty reported cases have received this therapy to date, it has become apparent that ACTH is not the panacea for patients with myasthenia gravis. Its use is not without danger but nevertheless some patients have been reported to have obtained definite improvement. A backlog of experience will be necessary before the final role of ACTH in the treatment of myasthenia gravis can be delineated. We have recently treated three patients with myasthenia gravis with ACTH, and it is felt that a report of our experience with these patients plus a review of the literature on this subject might prove of interest.

Review of the Literature

In 1948 Soffer et al[5] reported that the administration of 400 milligrams of ACTH to a patient with myasthenia gravis, with a thymic tumor, resulted in a marked decrease in the size of the tumor, a marked improvement in the clinical symptoms, and elimination of the daily necessity for prostigmine. In 1949 Torda and Wolff[6] administered a course of 400 milligrams of ACTH to each of five patients moderately to severely ill with myasthenia gravis. These patients during the three years before their study began had experienced minor transient fluctuations but no long-lasting nor significant changes in their clinical states. During the period of administration the patients experienced an increase in disability. The second day after completion of the series of injections the patients experienced changes suggesting the beginnings of an incomplete remission of the disorder. This consisted of marked improvement of muscle function while on appreciably reduced neostigmine bromide. Subsequently Torda and Wolff[7,8] reported further on their experiences with ACTH in myasthenia gravis. In their last report they reviewed the effect of ACTH on fifteen patients with this condition. Five patients received two courses of ACTH and two patients received three courses of ACTH. Ten patients exhibited a significant partial remission, and for four the results were inconclusive. One of the patients with so-called bulbar manifestations died on the third day of administration of ACTH. A new series of ACTH induced further remission in all patients to whom it was administered. Millikan and Eaton[2] administered ACTH and cortisone to five patients with myasthenia gravis. There was a history of long-term myasthenia without remission in every instance, and in two of them the disease was gradually becoming more severe. Two of the patients received three and two courses of ACTH ranging in dosage from 800 milligrams to 1,500 milligrams per course. Three of the patients received cortisone therapy in total doses ranging from 1,300 to 1,500 milligrams. One patient who received cortisone needed respirator care and tracheotomy three days after the completion of the course and subsequently succumbed eight days later. In three of the patients transitory improvement was observed. Shy et al[4] treated one myasthenic with cortisone. This patient was

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made worse by cortisone but returned to his previous status on the discontinuance of treatment.

Case Reports

Case 1.—The onset of myasthenia gravis in this white woman, born in 1922, was in January, 1944, when she developed ptosis of the right upper lid. Subsequently, she developed dysarthria, dysphagia, easy fatigability, and difficulty with mastication. Her myasthenia remained mild and was controlled on 30 to 75 milligrams of prostigmine hydrobromide per day. During her second pregnancy, commencing in October, 1946, and for four months after delivery, she required no prostigmine. Gradually her myasthenia gravis increased in severity and 345 milligrams of prostigmine hydrobromide per day were required to control her symptoms. In April, 1949, it was necessary to impose some limitation of her activities and in spite of this the patient commenced to lose weight. An acute exacerbation of her condition was precipitated in April, 1949, by an upper respiratory infection, and hospitalization was necessary at this time, during which the patient received intramuscular prostigmine. The patient was again hospitalized on June 16, 1949, for regulation of her medication following an episode of diarrhea and vomiting. On discharge from the hospital the patient was receiving 270 milligrams of prostigmine per day plus ⅜ of a grain of ephedrine three times a day. During the month of September her prostigmine requirements increased to 375 milligrams of prostigmine hydrobromide per day, and it was necessary to markedly restrict her activities.

Her third admission to the University Hospitals was in September, 1949, and this was precipitated by an upper respiratory infection. Examination on admission to the hospital at this time revealed an acutely ill patient. There was a marked bilateral ptosis, a marked dysarthria, and all the ocular muscles were weak, especially the right internal rectus and the right inferior rectus. A weakness of the palate and tongue could be demonstrated. There was a weakness of all the facial muscles, and a slight diaphragmatic weakness was present. Intramuscular and oral prostigmine failed to control her symptoms and consequently intravenous administration of prostigmine was commenced on her second hospital day. The prostigmine methylsulfate was dripped in at the rate of .5 mg. (1 ampoule) per hour. The patient felt comfortable in this regime. About two hours after the onset of the intravenous administration the patient choked while cleaning her teeth, and following this there was an excessive outpouring of mucus. The rate of intravenous administration was increased, and as the patient complained of flushing, headache, and intestinal discomfort, the intravenous was temporarily discontinued. Four minutes later the patient experienced great difficulty in breathing, and while being placed in a mechanical respirator, her respiration stopped completely and she became comatose. Seconds after the institution of mechanical respiration she regained consciousness and her color improved. One milligram of prostigmine methylsulfate was given intramuscularly, and the patient once more became comfortable. A nasal tube was passed in order to aid in the nutrition of the patient. During the next twelve-hour period her prostigmine requirements were .5 mg. of prostigmine intramuscularly every hour, and three doses of gr. 1/100 atropine were injected to control the side effects of the prostigmine.

On the evening of September 19, the patient's condition continued to deteriorate. Her dysarthria and dysphagia were marked and frequent suctioning was necessary as the secretions continued to be a problem. The patient was able to breathe only for short periods without the respirator and her prostigmine requirements became .5 mg. per hour via the intravenous drip and .5 mg. intramuscularly per hour. On the morning of her third hospital day the patient had a sudden outpouring of secretions, became unable to breathe, and lapsed into coma. Within a matter of a minute and a half the patient was intubated and oxygen administered, and the patient regained consciousness. A tracheotomy was then performed, and this was followed by bronchoscopy, by means of which considerable mucus was removed from the left lower bronchus. On the afternoon of the third hospital day ACTH administration was commenced at six-hour intervals and a total of 100 milligrams was given during the next thirty-hour period. As the patient's immediate course did not appear to be benefited, the ACTH was discontinued at the end of the thirty hours.

The patient's condition continued to deteriorate. She became increasingly restless, and on the evening of the fourth hospital day her prostigmine requirements had reached .6 mg. per hour via the intravenous route and .5 mg. of prostigmine per hour intramuscularly. After several hours of this increased rate of prostigmine administration the patient's restlessness increased and she became psychotic. She complained of claustrophobia and developed auditory and visual hallucinations. She ripped out her intravenous needle and was without intravenous prostigmine for a period of two and one-half hours. Ten minutes following an injection of .5 mg. of prostigmine intramuscularly, muscular twitchings developed, the patient became comatose and failed to respond to painful stimuli. The prostigmine requirements were adjusted to one-half ampoule every half hour and the patient regained consciousness. Her psychotic behavior continued, and after consultation with Dr. I. M. Eaton it was decided that the patient had become prostigmine-fast and that she was now developing toxic symptoms from the prostigmine. The prostigmine therapy was discontinued and for the next twelve days the patient received only ephedrine, 20 milligrams, three times daily.

The patient in the respirator received the same type of care as is administered to patients with bulbar poliomyelitis which has been described in a previous publication.[3] One gram of potassium chloride was added to her intravenous feedings daily. The patient's condition gradually improved and she was able to stay out of the respirator for longer periods. Her degree of oxygenation was followed by means of the oxymeter. On the sixteenth hospital day oxygen therapy was discontinued. On the eighteenth hospital day the patient was removed from the respirator and prostigmine therapy was recommenced. Her dosage was adjusted and it was possible to keep her comfortable on 90 milligrams

of prostigmine hydrobromide per day and she was discharged from the hospital.

At home the patient was maintained as a bed patient. She remained comfortable and gained weight. This partial remission persisted until December, 1950, when her prostigmine requirements once more commenced to climb, and it was felt that the patient once more was going into an exacerbation of her disease. She was admitted March 22, 1950, to the University Hospitals for an elective course of ACTH therapy. At the time of her admission she was taking 255 mg. of prostigmine per day and three tablets of ephedrine, grains ⅜, daily.

ACTH therapy was commenced at 20 milligrams every six hours. Concommitant with the ACTH administration the patient became restless and developed insomnia. On the third day of ACTH administration the dosage was reduced to 20 milligrams twice a day. On the fourth day of ACTH therapy one-half hour after an injection of ACTH the patient complained of shortness of breath, difficulty in breathing, and pooling of secretions. Her condition continued to deteriorate and on the fifth day of ACTH therapy it was necessary to place the patient in a respirator and the ACTH was discontinued. A total dosage of 280 milligrams was administered. Coincident with the worsening of her clinical condition her prostigmine requirements increased. Following the discontinuance of the ACTH the patient commenced to improve and she went into an amazing partial remission and at the time of her discharge on March 30, 1950, her prostigmine requirements had dropped to 195 mg. per day with a definite improvement in her strength and general condition. The patient continued to improve, and when seen in April, 1951, her prostigmine requirements had dropped to between 80 and 120 mg. of prostigmine hydrobromide per day. At this dosage she was able to carry on normal activities and compared her condition to the way she had felt during the remissions associated with her second and third pregnancies. On neurological examination the only abnormalities which could be demonstrated were bilateral paresis of the orbicularis oculi and slight paresis in the left upper extremity.

Comment.—This patient demonstrates the difficulties in handling an acute exacerbation of myasthenia gravis. The administration of ACTH during an acute phase of her disease appeared to cause a worsening of her condition. Subsequently a partial remission developed. Following a second course of ACTH there was a definite improvement of the patient's condition, and she entered an amazing remission of her disease which has persisted for a one-year period.

Case 2.—This white woman, aged twenty-seven, had the diagnosis of myasthenia gravis established in 1948 following the insidious onset of progressive muscular fatigue, weakness of grip, dysphagia, and episodes of diplopia and ptosis. She responded well to prostigmine therapy at this time. Shortly after the diagnosis had been established, the patient became pregnant, and commencing at the fourth month of her pregnancy and persisting to three months post-partum, the patient entered a marked remission of her disease with a sharp decrease in the amount of prostigmine which she required. However, the muscular fatigue and dysphagia again became more pronounced and the dosage of her prostigmine was gradually increased until she was taking 240 mg. of prostigmine hydrobromide and ⅜ grains of ephedrine three times daily without relief of her symptoms.

Her condition remained about the same until a "boil" was lanced in her right ear. Following this her symptoms increased in severity and she was admitted to the University Hospitals on December 10, 1950. At the time of this admission to the hospital she complained of difficulty in breathing. There was paresis of the right medial rectus muscle. There was a nasal quality to the voice. The soft palate moved only slightly. There was a mild bilateral weakness of the tongue muscles. She weighed 88 pounds, having lost 22 pounds since the onset of her illness. One day following her admission to the hospital the patient suddenly developed severe respiratory difficulty and inability to handle her secretions. She was placed in a respirator and a tracheotomy performed. Her prostigmine requirements increased until she was receiving .5 mg. prostigmine methylsulfate intramuscularly every two hours. In spite of the increased dosage of prostigmine the patient's condition failed to improve and her secretions of mucus were excessive. It was felt that the patient was prostigmine-fast and that the excessive secretions of mucus might represent a toxicity to the prostigmine. All prostigmine was discontinued and for ten days she received only 25 mg. of benadryl daily. On the eighteenth hospital day .5 mg. of prostigmine methylsulfate every two hours was recommenced, and the next day increased to 1.5 cc. every two hours. At the same time ephedrine 25 mg. three times a day was administered and atropine grains 1/200 was used as often as every four hours for control of the excessive mucus. The patient gradually improved and was able to stay out of the respirator longer on progressively smaller doses of prostigmine. On the forty-sixth hospital day the patient was switched to oral prostigmine. She was discharged on 180 mg. of prostigmine hydrobromide per day. At this dosage she felt "almost normal."

The patient was readmitted to the University Hospitals on February 21, 1951, for an elective course of ACTH therapy. The ACTH was administered in the hope it might prolong her remission. At the time of her admission to the hospital she was in a striking remission of her disease. No detectable muscle weakness could be demonstrated. She had gained 20 pounds and had been doing all her work. Her prostigmine requirements were 240 mg. of prostigmine hydrobromide per day. Following her admission to the hospital and the institution of bed rest, it was possible to reduce her dosage of prostigmine to 150 mg. per day. The patient was started on 20 mg. of ACTH therapy on the first day. This was increased 20 mg. per day until she was receiving 20 mg. every six hours. The total amount of ACTH administered was 380 mg. There was

no evidence of any reaction to ACTH therapy at any time, and it was possible while she was receiving the ACTH to reduce her prostigmine requirements to 120 mg. per day. She was discharged on March 6, 1951.

When the patient was rechecked in the out-patient department on April 10, 1951, her condition was essentially unchanged. She found that she felt best on 180 mg. of prostigmine daily and ⅛ grain ephedrine three times a day. The patient commenced taking ephedrine one week prior to her out-patient visit and found that without the ephedrine she experienced fatigability and difficulty in eating.

Comment.—This patient's myasthenia gravis progressively became severe until she developed an acute exacerbation of her disease. Tracheotomy and respirator care were necessary. The patient became prostigmine fast. Her condition remained exceedingly precarious for nearly a month and at the end of this time she apparently began a spontaneous remission. The ACTH was administered when the patient was in a partial remission of her disease and at the time of this report had produced no appreciable effect on her condition. The role of ACTH in prolonging her remission will be difficult to determine.

Case 3.—This white woman, aged thirty-five, dated the onset for her illness to December, 1950, at which time she noticed that she was having difficulty walking. Her muscle weakness increased, and the diagnosis of myasthenia gravis was established at the University Hospitals on January 25, 1951. The patient was hospitalized for the regulation of her prostigmine requirements and was discharged improved on January 29, 1951. Shortly after her return home she commenced to have cyanotic episodes precipitated by emotional stress. In addition she began to have progressive difficulty in coughing up her tracheal secretions.

The patient was readmitted to the University Hospitals on May 10, 1951. Examination at this time revealed an extremely apprehensive patient who was seriously ill. Neurological examination revealed paresis of the third, fifth, seventh, ninth, tenth, eleventh and twelfth cranial nerves. There was a generalized muscle weakness. The patient was having difficulty breathing and the movements of the chest and diaphragm were decreased. The accessory muscles of respiration were being used. In spite of increased prostigmine administration, the patient continued to lose ground, and twenty-four hours after her admission an emergency tracheotomy was performed and the patient was placed in a mechanical respirator. On the third hospital day it was decided that the patient was prostigmine-fast, and consequently this drug was discontinued. On the tenth hospital day another trial of prostigmine therapy was attempted but due to the unfavorable response was discontinued after twenty-four hours. On the eleventh hospital day the patient developed pulmonary atelectasis, which was partially relieved by bronchoscopy. The patient failed to show any improvement and was maintained solely by the aid of the respirator and by good nursing care. It was felt that if something were not done to alter her course she would succumb, and ACTH therapy was recommended.

On her twenty-eighth hospital day ACTH therapy was commenced. During the next thirteen days she received 940 mg. of ACTH administered in 20 mg. doses. On the fortieth hospital day prostigmine therapy was recommenced without materially affecting her symptoms. On the forty-first day she suddenly became cyanotic and lost consciousness. The patient was bronchoscoped and a mucous plug removed. However, she failed to regain consciousness and expired on this forty-first hospital day.

Comment.—The patient was following a progressively downhill course at the time ACTH administration was commenced. The ACTH did not alter the course of her disease and the patient succumbed on the thirteenth day of ACTH therapy. At post-mortem she had bilateral bronchial pneumonia and a substernal thymoma.

Discussion

The exact action of adrenocorticotrophic hormone in patients with myasthenia gravis remains obscure. Torda and Wolff[8] have attempted to shed some light on this problem and present interesting data which indicates that the immediate cause of the symptoms of myasthenia gravis is a decrease of acetylcholine synthesis and the administration of ACTH increases acetylcholine synthesis *in vivo*. Further studies of this sort will be necessary before the mode of action of ACTH in patients with myasthenia gravis is completely understood.

The results of administration of ACTH to the three cases documented here parallel the results previously reported by others. In the first patient reported there was marked improvement, in the second no change, and the third patient succumbed while ACTH was being administered. The definite increase in muscle weakness necessitating respirator care in the first patient following the administration of 280 milligrams of ACTH serves to underwrite the danger of ACTH administration and the necessity for having available all facilities to handle a crisis. The suggestion of Torda and Wolff[8] that the severity of the symptoms during administration of ACTH seemed to be reduced by the concurrent daily administration of from 3 to 5 grams of potassium chloride by mouth during the period of administration of

ACTH and for one day thereafter is worth further trial.

The spontaneous remissions in the first and second patients reported they had been critically ill and no longer responded to prostigmine, stress the necessity for continuing to energetically treat all patients even though at times the outlook appears hopeless. The mechanism of such spontaneous remissions is not understood. It may be that the stress to which a patient is subjected during a crisis serves to cause an increased production of ACTH which is eventually a factor in inducing a remission. The effect of administering the first course of ACTH in the first patient is difficult to assess since she was in such poor condition at the time of this administration. However, it is our impression that the ACTH temporarily caused a worsening of her state. Our experience in administering ACTH to the first patient and to the third one, who was acutely ill and succumbed while receiving the adrenocorticotrophic hormone, leads us to agree with Torda and Wolff[7] that ACTH is not the medication of choice in patients who are moribund. Rather, it would appear best to rely on the conventional methods of treatment in the hope that a spontaneous remission (as in the second patient) will eventually appear.

The use of ACTH during a remission of myasthenia gravis in the hope of prolonging the remission (as in the second patient) is of doubtful value. In view of the natural fluctuations in the course of myasthenia gravis, many years of study would be necessary to determine the worth of this procedure. In the light of our present knowledge, it is questionable if patients with myasthenia gravis should be subjected to the dangers of ACTH therapy when clinically they are well. Such a procedure must be considered purely experimental.

The induction of the dramatic remission following the second course of ACTH in the first patient is in accord with the observation of Torda and Wolff[8] that repeated small courses of ACTH administration may be necessary.

It has become obvious from the reported experience with ACTH that this hormone is not a cure for myasthenia gravis. However, in some cases it has induced gratifying remissions. In the group of patients whose clinical condition is deteriorating in spite of adequate therapy, and in the group whose condition has become static with marked limitations of their daily activities, a course of ACTH deserves serious consideration.

Conclusion

1. A review of the literature on the use of ACTH in the treatment of myasthenia gravis has been recorded.

2. Three cases of myasthenia gravis treated with ACTH are reported. In one case an amazing remission of the disease followed the termination of the second course of treatment.

3. Because of the exacerbation of symptoms encountered in some patients receiving ACTH therapy, this therapy should be restricted to centers equipped to treat an acute exacerbation of the disease.

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UNRESOLVED PNEUMONIA

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THE literature is rather voluminous on the sequelae of pneumonia but relatively scant concerning unresolved pneumonia. The common use of such synonyms as: interstitial, chronic, fibrous, fibrosing, organizing, protracted, indurative or persistent pneumonia, illustrates how loosely the condition is labeled. These terms are often used to designate chronic pulmonary conditions without attempt to determine the underlying cause.

The purpose of this paper is to review the necropsy cases of unresolved pneumonia at Saint Luke's Hospital over a thirteen-year period from 1937 to 1950 inclusive. It is hoped that such a review will stimulate more thorough investigation as to the underlying pathology and thus avoid an erroneous diagnosis and an opportunity for a cure of a remedial condition.

Incidence

The reported incidence of from 2 to 6.3 per cent[10,17] depends on whether the series are based on clinical diagnosis or necropsy findings. In 125 necropsy cases of lobar pneumonia at the Bellevue Hospital over a two and one-half year period, non-resolution was found in four, or 3.2 per cent of cases.[22] At the Russell Sage Institute, ten (6.3 per cent) of 159 necropsy cases of lobar pneumonia were so diagnosed on pathologic examination.[17] In two series[14,24] totalling twenty cases, the ages ranged from twelve to sixty-seven years with predominance of males.

At this institution, out of a total of 2,538 necropsies, exclusive of live and stillbirths, unresolved pneumonia was encountered in twenty-two or about 0.9 per cent of cases. The age ranged from three and one-half months to ninety years. There were seventeen males and five females, for a ratio of about 3.5 to 1.

Etiology

The bacteriologic agents in pneumonia are not primarily implicated with the factors responsible for non-resolution.[1] There are a variety of underlying major etiologic factors and the term unresolved pneumonia is therefore often a clinical one and not an expression of a specific pathologic entity or etiology.[5] The majority of cases have their origin in some other underlying pathology.[2,5,6,10,13,15,18,19,20,23,25]

In a series[5] of fifty-two cases diagnosed as unresolved pneumonia, impaired bronchial drainage of varied etiology accounted for twenty-two (42.3 per cent) with four (7.7 per cent) due to bronchogenic carcinoma. The same series revealed twelve (23 per cent) due to some intrinsic complication of pneumonia such as: lung abscess, atelectasis, pleural effusion or empyema. Bacteriologic and chemical agents such as Klebsiella pneumoniae, oil aspiration, streptococcus and aspiration of anerobic organisms of the oropharynx, accounted for sixteen (30.7 per cent) of the cases. In two (3.8 per cent), the major etiologic processes could not be determined.

The delay or failure of the usual process of resolution to take place, has been attributed to either defective phagocytic action of the leukocytes or failure of them to liberate an autolytic enzyme.[4,11,17] Influenzal, streptococcal and Friedlanders pneumonia as well as the broncho-pneumonia accompanying measles and pertussis are most likely to result in chronic pulmonary sequelae.[8]

Syphilis has been reported as predisposing to delayed or non-resolution of lobar pneumonia.[8,23,25] In a series of thirty patients with non-resolution, pulmonary abscess and bronchiectasis, twelve (40 per cent) occurred in association with proved pre-existing syphilis.[23] This may be purely coincidental since pulmonary fibrosis due to syphilis is extremely rare.

In our series (Table I) impaired bronchial drainage as a major underlying factor, accounted for eleven (50 per cent). Intrinsic complications accounted for three or approximately 14 per cent, and three or another 14 per cent were of undetermined origin. Five (22.7 per cent) had a history of three or more attacks of "so-called" pneumonia.

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TABLE I. MAJOR UNDERLYING FACTORS

Underlying Factors	Number of Cases	Percentage
Impaired Bronchial Drainage		
Bronchiectasis	4	18.2
Chronic bronchitis	2	9.0
Tuberculosis	2	9.0
Bronchial asthma	2	9.0
Bronchogenic carcinoma	1	4.5
Intrinsic Complications		
Empyema	2	9.0
Pulmonary abscess	1	4.5
Undetermined	3	13.5
Previous History of Repeated Pneumonia	5	22.7

TABLE II. SYMPTOMS

Symptoms	Number of Cases	Percentage
Cough	19	86.3
Shortness of breath	14	63.6
Chills	9	40.9
Fever	8	36.3
Chest pain	7	31.8
Weakness	7	31.8
Hemoptysis	6	27.3
Previous pneumonia	5	22.7
Weight loss	4	18.0
No symptoms	2	9.0

Bronchiectasis is likewise often a clinical diagnosis. Although four (18 per cent) of our cases revealed bronchiectasis at necropsy, there is some question as to whether it is an antecedent process or the result of the compensatory emphysema and or pulmonary fibrosis.

Symptoms

Unresolved pneumonia is characterized by its subacute or chronic clinical course.[24] The symptoms usually consist of productive cough, shortness of breath, chills and fever, generalized weakness, chest pain, hemoptysis, weight loss and history of previous episodes of pneumonia.[7,12,14,15,24] In a series of ten cases,[14] a productive cough of mucopurulent sputum occurred in all cases; hemoptysis in eight; chest pain and fever in five cases each. The duration of symptoms varied from six months to twenty-two years, with 70 per cent between two and ten years duration. In another series of ten cases,[24] the duration of cough, fever, expectoration, hemoptysis and chest pain varied from two and one-half months to five years.

In our series (Table II) a productive cough occurred in nineteen (86.3 per cent); shortness of breath in fourteen (63.6 per cent); chills in nine (40.9 per cent); fever in eight (36.3 per cent); chest pain and generalized weakness in seven (31.8 per cent); hemoptysis in six (27.3 per cent) and weight loss in four (18 per cent). In five cases (22.7 per cent) a history of repeated attacks of pneumonia was elicited. In a series of 220 cases of pulmonary fibrosis, 163 (74 per cent) were traced to previous attacks of pneumonia, influenza, measles or pertussis.[8] No definite subjective symptoms could be elicited in two (9 per cent) of our cases.

The duration of symptoms is illustrated in Table III. The symptoms of cough, shortness of breath, chest pain, weakness and hemoptysis were present from one to five years in 95 per cent of the cases.

The symptomatology in general is one of repeated episodes of respiratory tract illness of varying severity.[12] Physical examination usually yields divergent findings and varies with the size and location of the disease process.[15]

Pathology

The basic pathologic changes are the result of the transformation of the intra-alveolar exudate into fibrous connective tissue.[4,17,22] The characteristic indurated leathery lung requires time plus some nidus of infection or irritant and occurs as a natural protective mechanism.[20]

The lung changes depends on the degree of injury to the various structural components. The pneumonias in which the connective tissue framework, bronchial tree, vascular and lymphatic channels are seriously damaged or destroyed, exhibit the highest number of chronic pulmonary sequelae.[8]

The pathologic physiology is primarily the result of interference with bronchial drainage and the resultant secondary changes of obstruction, such as pneumonitis, collapse, bronchiolitis, bronchiectasis, abscess and gangrene.[15] Emphysema, which impairs respiratory function, develops to accommodate for the loss of efficiency of the fibrotic lung.[8] Improper ventilation and drainage plus impaired peristaltic action of the cilia results in the puddling of secretions and inadequate cleansing of the involved bronchopulmonary segment.[5] The metaplastic transformation of the ciliated epithelium may further add to stagnation of secretions. These may be the necessary nidus of infection or irritant to stimulate the natural protective mechanism of reactive fibrosis.

The affected lung is rubbery to leathery in consistency and airless. The pleura may or may not be thickened. As a rule whole lobes or at least large areas are involved but patchy distribution has been reported.[17,21] In a total of thirty cases[14,17,23] the right upper lobe was involved in six (20 per cent)

TABLE III. DURATION OF SYMPTOMS

Symptoms	Range	Duration: Under 6 months	Under 1 year	1-5 years	5-10 years	10-15 years	15-20 years	Over 20 years
Cough	3 weeks to 30 years	2	4	5	3	2	0	3
Shortness of breath	6 months to 30 years	0	3	6	2	2	0	1
Chest pain	6 months to 6 years	0	0	4	2	0	0	0
Weakness	3 weeks to 8 years	1	0	4	1	0	0	0
Hemoptysis	2 months to 20 years	1	0	2	0	1	0	1

of cases. The right middle and lower lobes accounted for five each (16.9 per cent). The right upper and middle, right middle and lower and the left lower lobe were involved in three cases each (10.0 per cent). All lobes of the right lung in two cases (6.9 per cent) and one case each (3.3 per cent) involved the left upper, both lower lobes, with the right middle lobe and patchy distribution of the other lobes of the right lung.

In our series (Table IV) involvement of all lobes of the right lung accounted for five (22.7 per cent) of the cases. The right lower, right upper and the middle lobe accounted for three cases each (13.6 per cent). In two cases each (9.0 per cent) the involvement was limited to the right upper, right middle and lower and the left lower lobe. All lobes of the right lung or singular involvement of the lobes occurred in 68 per cent of the cases. The remainder of five cases revealed a variance in their distribution.

The histopathology is one of interstitial fibrosis, thickening of the alveolar walls, lymphocytic and neutrophilic infiltration, lymphocytic hyperplasia, atelectasis and vacuolated macrophages in alveolar spaces. The foregoing are basically the result of an obstructive pneumonitis. The intra-alveolar exudate of any pneumonic process with its neutrophils and fibrin may resolve and be absorbed. This same process may progress into suppuration with abscess formation and necrosis. The exudate may undergo fibrinoid changes with ingrowths of fibroblasts and transformation of the fibrinous plugs into fibrillar connective tissue. The basic distinctive histology of the lung is thus replaced by a chronic inflammatory process.[12,22] The reactive fibrosis is usually most marked in and about the bronchial segments and the alveolar walls. This process leads to the distortion of bronchioles and alveoli with areas of obstructive pneumonitis and resultant atelectasis, compensatory emphysema, bronchiectasis and obliterative bronchiolitis. The pooling of secretions result in interstitial suppuration, abscess formation, purulent bronchitis and even necrosis of lung tissue with gangrene as the result of vascular thrombosis.[8]

TABLE IV. LOCATION OF PATHOLOGY

Location	Number of Cases	Percentage
All lobes, right lung	5	22.7
Right lower lobe	3	13.6
Right upper and middle lobe	3	13.6
Right upper lobe	2	9.0
Right middle and lower lobe	2	9.0
Left lower lobe	2	9.0
Left upper and lower lobe	1	4.5
Lower lobes, diffuse	1	4.5
Lower lobes, patchy	1	4.5
Right middle and lower and left lower lobe	1	4.5
Right lobes and left lower lobe	1	4.5

A review of the histopathology in our series revealed no satisfactory criteria for an adequate classification. The cases of relatively short duration revealed a histologic picture almost identical with those of considerable longer duration and consisted of replacement of lung tissue by a fibroblastic and hyalinization process, obliteration of air spaces, thickening and distortion of bronchioles, abscesses, areas of necrosis and emphysema with lymphocytic and neutrophilic infiltration in bronchioles, alveoli and interstitial tissue. A careful search failed to reveal any evidence of oil aspiration. In general, our post-mortem bacteriology was poor and therefore offered no accurate bacteriologic etiology.

Diagnosis

The usual symptoms of productive cough, shortness of breath, chills and fever, chest pain, generalized weakness and hemoptysis, may be characteristic of a variety of pulmonary disease processes.

The use of modern antibiotics has reduced the mortality in pneumonia with rather resultant dramatic clinical improvement. The pathologic and physiologic changes may not always keep pace with the clinical improvement and thus the masking effect of the antibiotics may give one a false sense of security.[9] A diagnosis of unresolved pneumonia without a thorough search for the

underlying factors may result in missing a remediable condition.[1]

In a series of forty patients diagnosed as atypical pneumonia, a pneumonitis around a preexisting bronchiectasis was found in 67.5 per cent of cases.[6]

Any pneumonia which does not improve clinically and radiologically in two to four weeks should have a complete tracheobronchial study with: (1) repeated serial chest x-rays, (2) bronchoscopy, (3) studies for malignant cells and acid-fast organisms and (4) bronchograms.[9,10,15] Bronchoscopy should be done early to avoid irreversible damage.[10,15] In a series of thirty-eight cases, diagnosed as unresolved pneumonia, bronchoscopic examination revealed eight bronchogenic carcinomas, five of bronchiectasis, two each due to foreign bodies and lung abscess, one each of extrabronchial tumor, inflammatory bronchial stenosis and enlarged bronchial glands. The remainder, eighteen children, revealed varying degrees of incomplete bronchial obstruction due to secretions.[15] In a series of fifty-two cases diagnosed clinically as unresolved pneumonia, the diagnosis was proved correct on necropsy examination in only six (11.5 per cent) of cases.[20]

In our series the correct clinical diagnosis was made in only three (13.6 per cent) of the cases. No bronchoscopic examinations had been made.

Treatment

The use of irradiation and/or diathermy has produced relief of symptoms and clearing as indicated by x-ray examination. The series, however, has been too small to draw any definite conclusions.[7,16]

As a result of the advances in anesthesia and thoracic surgery, cures of inflammatory lesions of the lung by surgical extirpation have been reported with increasing frequency during recent years.[24]

Conclusions

1. The use of such a vague term as unresolved pneumonia is in reality a diagnosis of an undiagnosed case. The more specific term of "obstructive pneumonitis" based on the underlying pathology is the one of choice.

2. Out of a total of 2,538 necropsies from 1937 to 1950 inclusive, twenty-two cases were encountered, for an incidence of 0.9 per cent.

3. Impaired bronchial drainage as the major underlying factor accounted for 50 per cent of the cases. Intrinsic complications accounted for 14 per cent and in another 14 per cent no definite factor could be determined.

4. A history of repeated attacks of so-called pneumonia was elicited in 22.7 per cent of cases.

5. The symptoms of a productive cough occurred in 86.3 per cent; shortness of breath in 63.6 per cent; chills in 40.9 per cent; fever in 36.3 per cent; chest pain and weakness in 31.8 per cent each and hemoptysis in 27.3 per cent. The symptoms of cough, shortness of breath, chest pain, weakness and hemoptysis were present from one to five years in 95 per cent of the cases.

6. Involvement of all the lobes of the right lung, singular involvement or combinations involving the right lung occurred in 75.6 per cent of cases.

7. The correct clinical diagnosis was made in only 13.6 per cent of cases.

8. A thorough and complete tracheobronchial study is imperative if an early diagnosis of this frequently remediable condition is to be made.

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(Continued on Page 1191)

A BOTANICAL CONSIDERATION OF THE WEED OLEORESIN PROBLEM

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THE problem of weed dermatitis in the State of Minnesota is by no means new. As early as 1894 MacDougal[6] published a case of dermatitis due to the showy lady's-slipper *(Cypripedium reginae, C. spectabile)* and the moccasin flower *(C. calceolus* var. *pubescens)*. He noted in addition that there were a number of native Minnesota plants which had been definitely ascertained to be more or less "poisonous" by means of skin contact. Among these he listed the following:

Cocklebur *(Xanthium canadense)*
Smartweed *(Polygonum hydropiper)*
Wood Nettle *(Laportea canadensis)*
Beggar's-Ticks *(Bidens frondosa)*
Skunk Cabbage *(Symplocarpus foetidus)*

It is interesting to note some of the author's further observations in the light of information since uncovered: "No doubt the larger percentage of the common plants known to be poisonous is due to the fact that they offer much more opportunity for observation than do the rarer forms. . . . It is by no means to be understood that there are many plants which are always and invariably poisonous, for many of the plants in this category are injurious to only a very small percentage of the persons touching them, so that their irritating qualities might remain undiscovered altogether unless tested by a large number." MacDougal reasoned that these noxious plants poisoned one by piercing glandular hairs or by means of volatile agents (e.g., toxicondendric acid of Rhus).

These early acute observations, together with those of Blackley, Walker, Hannah[5] and others have since been much extended and clarified by: (1) the immunologic concepts of Bloch and Jadassohn, (2) the chemical and immuno-biologic studies of Coca, Brown, Milford[1] and others, (3) the clinical studies of Shelmire,[8] Brunsting,[2] Sulzberger,[9] Anderson[2] and others; so that as a consequence of this substrate of information, it is pretty well established that in a gross qualitative manner, at least, plants and their pollens may be separated into two fractions of clinical importance:

1. The water-soluble protein fraction relating to atopic manifestation such as hayfever, asthma, et cetera.
2. The organic solvent fraction relating to contact dermatitis.

During the past several years we have had the opportunity of studying twenty-five cases of eczema in which positive patch tests were obtained to the oleoresinous fractions of a number of weeds. In all of these cases there was present reasonable evidence that the positive skin test reactions were related to the patients' clinical condition.

Having obtained such information, one is confronted immediately with several problems: which weeds most frequently cause dermatitis, and which weeds giving positive skin reaction are present in a given patient's locality? Furthermore, inasmuch as positive patch test reactions in a given individual often occur within a group of weeds closely related to one another botanically, the question of their allergenic interrelationships appears. It would be extremely helpful, for example, to know the major and minor allergenic relationships of these weeds because such information could be employed in determining the constituency, proportion-wise, of the various weed oleoresins, which would be included in hyposensitization material to be administered to a given patient.

In regard to the geographic distribution of weeds of "allergic" importance in the state of Minnesota we have had the good fortune of having at our disposal previous studies of Ellis and Rosendahl[3] and of Rosendahl, Ellis and Dahl.[7] Having determined the weeds which appeared "significant" on the basis of our cases, such weeds were then plotted in terms of their geographical location in the state of Minnesota, as determined by the previously noted studies, in Figures 1 and 2. The utility of such data is quite evident. We are indebted to Dr. J. W. Moore for the collection sites of various species.

It is with considerable temerity that one ap-

Rhus oleoresins were omitted in this study.

From the Division of Dermatology, University of Minnesota, Director, H. E. Michelson, M.D.; the Department of Botany, University of Minnesota, Chairman, A. O. Dahl, Ph.D.; and the Veterans Administration Outpatient Clinic, Ft. Snelling, Minnesota, Chief Medical Officer, Richard B. Hullsiek, M.D.

proaches the question of the allergenic relationship of the various weeds. True, the chemistry of some plant material has been studied in considerable detail, e.g., rhus, primula, et cetera. But to producing dermatitis of the contact allergic type. Despite the previously noted information differentiating the water-soluble protein fraction from the oleoresinous fraction of weeds, both on

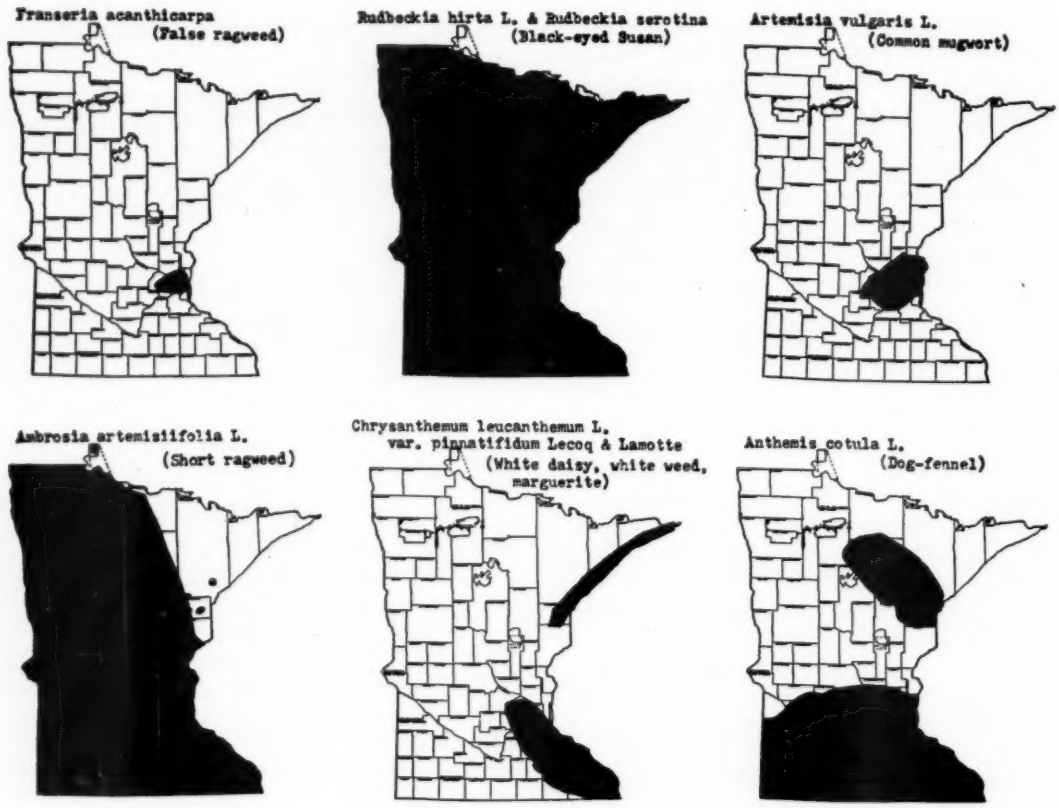

Fig. 1.

our knowledge information is rather sparse regarding the composition of the materials under consideration here either in a chemical or an immuno-biological sense.

The original confusion as to whether it was the pollen or other plant parts that were capable of producing the dermatitis has now been settled in favor of both. Simpson's early impression[10] that the weed dermatitis active principle was extractable in a number of organic solvents has since received support from Coca, Milford and Brown. These later investigators, by means of simple lucid experiments, demonstrated the gross chemical and immuno-biological differentiation between the water-soluble protein fraction capable of producing hay fever and asthma and the oleoresin, non-volatile organic extract fraction capable of a chemical and a biological (immunological) basis, it is a fairly common practice to attempt to transfer the concept of major and minor allergens as elucidated with respect to atopic states (hay fever, asthma), to the contact allergic state induced by oleoresins as well. For example, short ragweed water-soluble protein fraction, for all practical purposes, protects anaphylactically against burweed-marsh-elder, ragweed, cocklebur, et cetera. Therefore, it has been reasoned erroneously that a parallel situation exists with respect to the dermatitis producing oleoresin fraction of these same weeds. However, such a conclusion is not justified. We do not mean to deny the possibility that the concept of major and minor allergen may not pertain to oleoresinous extracts, but this is as yet unproven. As a starting point in the general

study of this problem, we decided to subject our data, and that of others, to a botanical analysis to determine what possible inter-relationships and trends might be determined by this device. Our insofar as tested, are more likely complexes of offending species, as illustrated in Figure 3.

Within the very real limits of the small population examined, it would appear from Figure 3

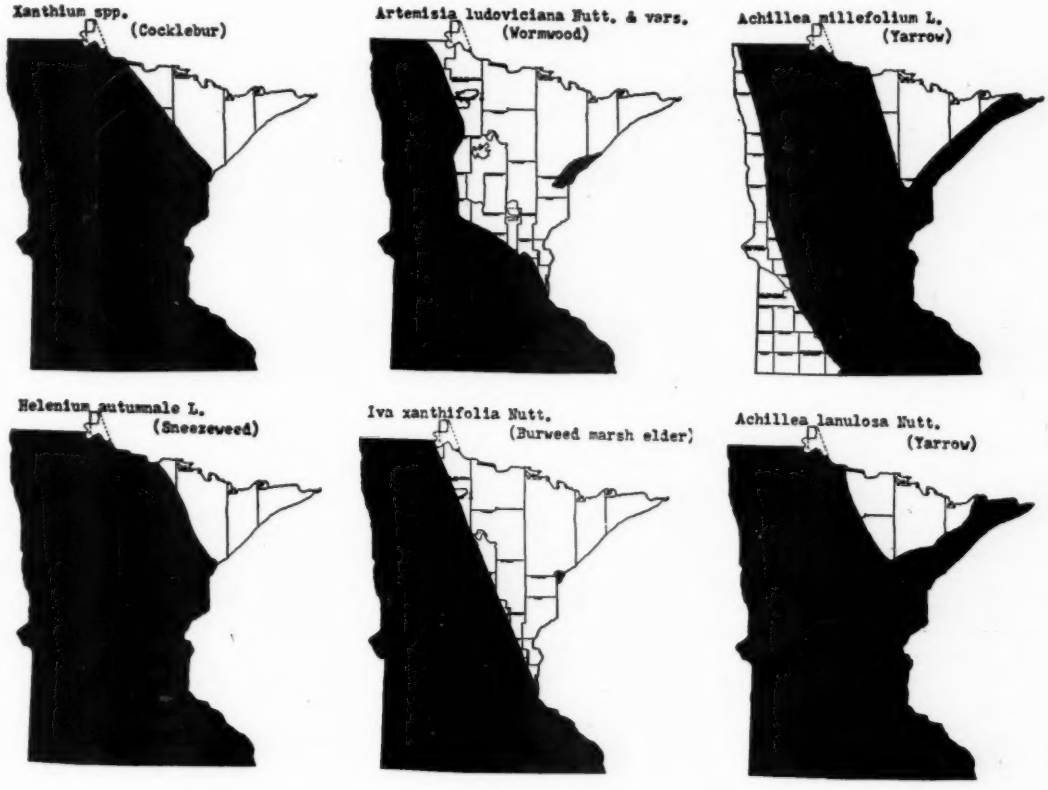

Fig. 2.

data include only those patients which showed positive patch test reactions to one or more weeds. The patients were tested with twenty-four commercial oleoresin weed extracts* in acetone. The material was applied to the upper back or abdomen in the usual manner. The positive cutaneous test reactions so obtained were plotted into a table arranged according to botanical relationships[4] (Table I). Technical names follow the treatment presented in Gray's *Manual*.[4]

These statistics corroborate our earlier clinical impression that the Family *Compositae* is an outstanding etiological agent in the production of weed contact eczemas. Further analysis reveals that certain tribes within the Family *Compositae*, that certain tribes within the *Compositae*, e.g., sunflower *(Helianthiae)*, dog fennel *(Anthemideae) and sneezeweed (Helenieae)* are the important sources of the sensitizing components.

It becomes apparent that just as there are differences in sensitizing potencies between tribes, so there are differences in sensitizing potencies within a given tribe (Fig. 4).

Considering the above data, it appears possible that short ragweed contains the major antigenic agent and shares major antigenic components with false ragweed, burweed-marsh elder and cocklebur. The antigenic relationship of these weeds to black-eyed Susan and sunflower, if there be any at all, must be a remote one. It is of some interest to note that also from a botanical point of view black-eyed Susan and sunflower are differentiated

*Graham Laboratories, Dallas, Texas.

TABLE I—Botanical Analysis

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	
Burweed Marsh Elder	+	+		+	+			+	+		+	+	+	+		+	+					+	+		+	Sunflower Tribe
Short Ragweed	+	+	+	+	+	+		+		+	+	+	+		+	+	+	+	+	+	+	+	+	+	+	
False Ragweed				?	+		+	+		+	+	+	+	+	+	+	+					+	+	+	+	
Cocklebur	+	+	+	+		+				+	+		+			+		+	+		+	+	+		+	
Black Eyed Susan																							+			
Sunflower																				+						
Sneezeweed		+		+		+		+	+	+	+	+	+	+	+	+	+	+	+	+	+		+	+	+	Sneezeweed Tribe
Yarrow													+		+		+			+						Dog Fennel Tribe
Dog Fennel				?			+							+	+						+	+				
Wild Feverfew	+		+	+	+		+	+	+		+	+	+	+		+	+					+		+	+	
Common Wormwood	+	+		+			+	+	+		+	+	+	+		+	+	+		+		+	+		+	
Smaller Burdock																					+					Thistle Tribe
American Thistle				+								+														
Dandelion															+		+									Chicory Tribe
Red Root Pigweed																	+									
Lamb's Quarters																										
Russian Thistle																										
Western Water Hemp																										
St. John's Wort															+			+								
Evening Primrose									+																	
Common Milkweed					?		+							+				+			+					
Horse Nettle					?																					
Golden Rod																										
Aster (MANY-FLOWERED)																										

from the four species just mentioned. For example, both black-eyed Susan *(Rudbeckia)* and sunflower *(Helianthus)* are insect-pollinated whereas the other species are solely wind-pollinated.

In Figure 5 we again note the disparity in reaction incidence within a botanical tribe. It would seem likely that common wormwood and wild feverfew are, on the basis of biological evidence (patch tests), probably linked closely together

antigenically, with the relationship to dog fennel and yarrow at best held by an antigen of a minor character.

Our number of cases is small and subject to criticism on that account. However, it is of interest and obviously supports our findings that Shelmire's study on weed dermatitis produces a profile very similar to our own (Fig. 6).

We have admittedly indulged in speculation beyond the bounds afforded by our information regarding what might represent major and minor allergenic relationships between and within the particular botanical groups discussed. Yet this was done with the intention of setting up hypotheses against which laboratory and clinical experiments may be pitted in the near future.

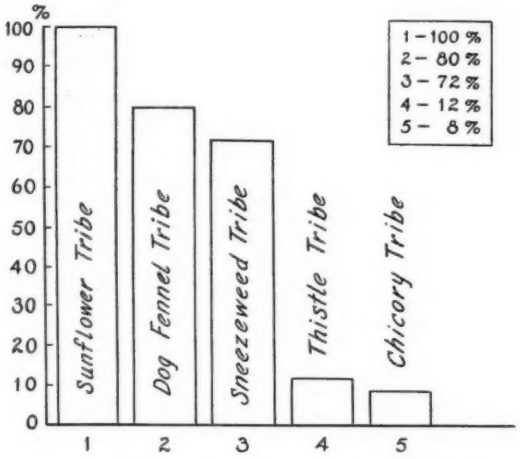

Fig. 3. Reactions within the Family *Compositae* (per cent).

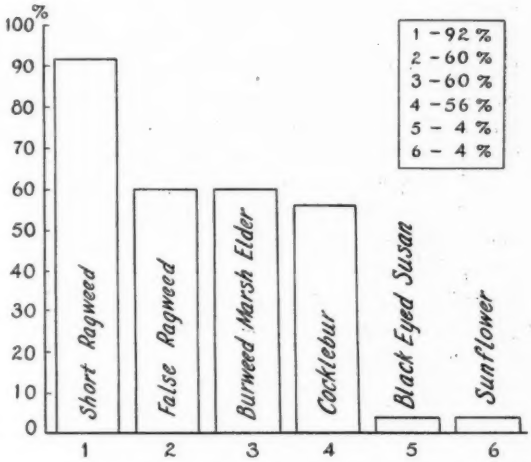

Fig. 4. Reactions within the sunflower tribe *(Heliantheae)* (per cent).

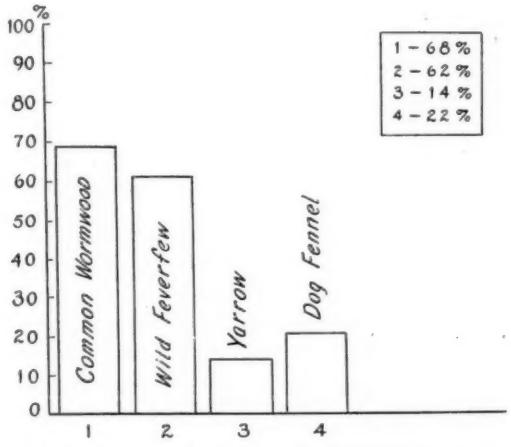

Fig. 5. Reactions within the dog fennel tribe *(Anthemideae)* (per cent).

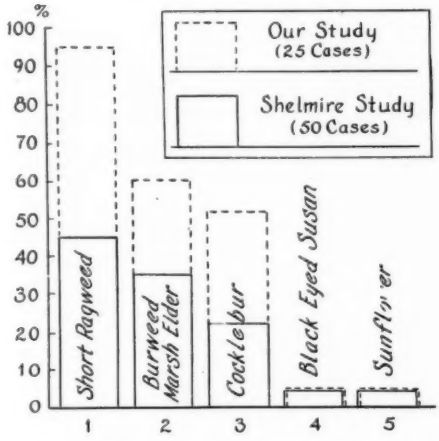

Fig. 6. Comparative studies: sunflower tribe *(Heliantheae)* (per cent).

Summary

1. The geographic distribution of weeds of dermatitic significance in the State of Minnesota has been illustrated graphically.

2. Analysis reveals that the large botanical Family *Compositae* contains the majority of offending weeds of those tested.

(Continued on Page 1188)

REPAIR OF VENTRAL HERNIA WITH KNITTED WIRE MESH

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THE use of metallic substances in the repair and reconstruction of various tissues has engaged the attention of surgeons for a great many years. The ancient Greeks used gold wire ligatures. The earliest efforts at tissue reconstruction with metal were made during the present era by dental surgeons and orthopedists, but in 1894 Phelps[15] described a method of placing silver wire loops between abdominal layers to cover large inguinal defects.

During the past decade surgeons have intensified their search for the perfect material to use in those frequently encountered cases where there are not sufficiently strong anatomical structures present for the successful repair of large ventral defects. The early disappointing results of the use of most metallic substances have been due to the electro-chemical phenomenon known as corrosion or oxidation of the buried metal. In the past few years with the development of fairly non-irritating and relatively inert alloys, this has been minimized. These metals resist corrosion by the formation of a thin film of an insoluble compound, usually an oxide, on their surfaces, preventing metal ions from migrating from these surfaces and also preventing the transfer of electrons from the metal to the oxidizing substances in the living tissues in which the metal is embedded.

Several metals, such as vitallium, tantalum and stainless steel, have this corrosion-resisting property and have been used surgically in various capacities with success. Vitallium, an alloy of cobalt, chromium, and molybdenum, which has been widely used in orthopedic surgery, has the disadvantage of being brittle, impossible to machine or draw into wire. Tantalum, the seventy-third element in the Periodic Table, was introduced to surgery by Burke in 1940.[3] It is highly resistant to oxidation in the tissues, has a tensile strength 50 to 80 per cent that of stainless steel, and can be stamped, machined, and drawn into wire. However, due to its low resistance to heat, it is difficult and costly to process. Stainless steel (18-8 alloy) is only slightly less resistant to corrosion; it is ductile, tough, and is generally available, costing only about one-sixteenth as much as tantalum. The alloy consists of 18 to 20 per cent of chromium, 8 to 10 per cent of nickel, 60 to 70 per cent of iron, with small amounts of carbon, manganese, silicon, and sulfur. It is a relatively poor conductor of heat, and in the soft annealed form used surgically it has a very high tensile strength and is somewhat more pliable than tantalum. In passivating, a thin protective film of chromium oxide forms upon the surface.

There have been a large number of reports of successful repair of ventral abdominal hernias with the use of tantalum gauze, made up of tantalum wire 0.003 inches in diameter, woven into a 50 mesh screen cloth, i.e., 50 wires criss-crossing to each square inch.[4,7,9,11,12,14,17,18] Numerous experimental studies as well as clinical observations have established that fibrous tissue grows through and around this mesh, forming, in a few months, a very strong plate of wire-reinforced scar tissue to cover the defect in the abdominal wall.[5,6,7,9,10] Babcock has buried wire screen made of very fine stainless steel between layers over weak areas of the abdominal wall for reinforcement and has found by his experiments on dogs that it is non-irritating and that in two weeks the wire is covered by granulation tissue grown through the interstices of the mesh. McNealy and Glassman, in May, 1950, devised a rigid vitallium prosthesis to be fitted into hernial defects much as a boot or shoe fits over a defect in an automobile tire.[13] The occasional use of non-metallic foreign materials such as nylon net[2] and preserved ox fascia[8] to bridge large defects in the abdominal wall has not been uniformly successful.

Hernias of the abdominal wall usually arise from a deficiency, either congenital or acquired from operation or other trauma, in the supporting structures, namely, the musculature or endoabdominal fascia, through which defect the peritoneum bulges as a hernial sac. Small and moderately large defects can be repaired successfully by various techniques using silk, wire, strips of fascia lata, pedicle flaps of fascia and muscle, split thickness skin grafts, et cetera, but the surgeon is frequently confronted with huge defects in the abdominal wall where his ingenuity is taxed by several problems. The size of the defect to be bridged may be such that it necessitates the use

of an extremely large amount of strong material. This is seldom available from the adjacent tissues since these are usually deficient or weakened by fat infiltration. There are some objections to the use of fascia: strips may be inadequate, and massive fascial transplants have poor lateral strength; moreover, obtaining a massive fascial sheet is not a simple operation and certainly adds to the operative morbidity. It is in these cases that a prosthesis of some sort is desirable. It should be strong, pliable, non-irritating in the tissues, workable, inexpensive, and easily available to the general surgeon in private practice.

The tantalum mesh previously described fulfills some of these desiderata. However, one serious disadvantage is apparent. Due to the inelasticity of this material, the outward force to which it is subjected from an increase in intra-abdominal pressure is transmitted in its entirety to the peripheral sutures anchoring the prosthesis to the surrounding tissues. This is of consequence especially during the early postoperative period when the tensile strength of the tissues is low. It has been frequently noted[14] that the gauze becomes more or less brittle and considerable fragmentation occurs, but this apparently does not affect the strength of the repair and the surrounding scar tissue seems to be dense enough to prevent migration of the free wires. The current cost of the tantalum mesh is about $25 for a 6 by 12-inch sheet.

Rudie[16] recently developed a crocheted tantalum fine wire mesh. This material, we believe, has more of the qualities of the perfect metallic substitute for an abdominal wall than any we have used heretofore. It has considerable elasticity and sufficient strength, yet has a comparatively small amount of metal to the square inch. The elasticity is such that during the early healing period outward force applied to it is largely dissipated before it reaches the peripheral holding sutures. After organization of the scar tissue infiltrating the interstices of the mesh, of course, the elasticity disappears.

Dr. Rudie kindly furnished us with several sheets of this material for our use in our early cases at Ancker Hospital, Saint Paul. Lately we have been using .005-inch (B&S gauge 36) stainless steel wire knitted with No. 3 knitting needles; this has the additional advantages of relative cheapness and easy availability to the surgeon in private practice.

The surgical technique in the use of the knitted wire mesh is very simple. After wide dissection and reduction of the hernia, the peritoneum is closed, by imbrication if it is redundant; the knitted wire sheet is then placed external to the peritoneum and sutured with interrupted fine wire sutures of the same material as that knitted, to the strong fascial tissues or bone, peripherally, the sutures being placed about ⅜-inch apart. The weaker fascial structures (there is rarely much muscle), which were overlying and immediately adjacent to the hernia, are laid over the mesh and sutured in place, covering the metal as completely as possible without tension. We have employed the use of small penrose drains, removing them after forty-eight hours, for with these extensive dissections uncovering wide areas hemostasis is rarely complete and considerable transudation occurs.

We have repaired nine very large ventral hernias with knitted wire mesh. Six of these were at Ancker Hospital, and three in private hospitals. This method of repair was reserved for those cases which we felt did not have sufficient tissue available for repair by any other means. This is apparent by the fact that during this period there were fifty-five postoperative ventral hernias repaired at Ancker Hospital by a variety of techniques. Suture materials used in these cases included silk, catgut, cotton, and fascia lata in strips and patches. There were four recurrences in this group, all occurring within six months of the repair.

In the nine repairs with knitted metallic wire, tantalum was used in three and stainless steel in the last six. Ages of the patients varied from thirty-five to seventy-six; five of them were over seventy years of age. In each of five patients one previous unsuccessful attempt at repair had been made; in one case, two attempts, in two cases, three attempts, and in one case, four attempts had been made before the final repair with metallic gauze. The primary operation in four cases had been cholecystectomy and choledochostomy; in two, gastric resection; in one, pelvic laparotomy; in one, combined abdomino-perineal resection of the rectum; and in one, nephrectomy.

To this date there have been no recurrences of hernia in these nine patients. Three of them were repaired more than two years ago; two between one and two years ago; two between six months and one year ago; and two less than six months

ago. In two patients we have noted a raised firm plaque exactly the size of the mesh used. There is no evidence of inflammation and no discomfort associated with this. In the other cases this plaque of reinforced scar is palpable but not visible.

Summary

Nine patients with extremely large recurrent postoperative ventral hernias have been repaired with knitted tantalum or stainless steel wire mesh laid in as a substitute for the attenuated muscle and fascia of the abdominal wall. The peculiar advantages of knitted wire mesh have been emphasized.

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CONGENITAL OBSTRUCTION OF THE ALIMENTARY CANAL IN THE NEWBORN

(Continued from page 1159)

Summary

Four proven cases of congenital obstruction of the alimentary canal in newborn infants have been reported, with one non-operative death. There were three cases of complete atresia, two of the esophagus and one of the duodenum, which occurred among 4,374 live-births in our hospital during the five-year period ending December 31, 1950.

The authors wish to emphasize the fact that congenital obstructions of the alimentary canal are more common than is generally recognized. Such obstructions are easily overlooked. Esophageal obstructions are frequently misdiagnosed "atelectasis" or "bronchopneumonia" because of the respiratory complications resulting from them. The symptoms of intestinal obstruction are frequently attributed to cerebral birth trauma or feeding problems until too late.

Every physician who is responsible for the care of newborn infants should be keenly interested in the problem of congenital obstruction of the alimentary canal. Excellent surgical results may be obtained when the diagnosis is established early, preferably within the first 48 to 72 hours of life.

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VENTRICULAR ANEURYSM

A Case of More than Twelve Years' Duration

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VENTRICULAR aneurysm, with few exceptions, was an unexpected finding at necropsy prior to the past two decades. During this period the time of diagnosis has changed from that of necropsy to clinical examination and today ante-mortem diagnosis is not an infrequent occurrence. A concomitant change has occurred in the literature on this subject.[1,3,7] The increased incidence of ante-mortem diagnosis of ventricular aneurysm has resulted from an increased index of suspicion by clinicians, and from increased use of electrocardiography and roentgenography. Although the presence of a ventricular aneurysm may be suspected during life from the history, physical examination and electrocardiogram, confirmation is dependent on roentgenography or exploratory thoracotomy.

The following report of a case is made because of the size of the aneurysm, its duration and the problem of its differential diagnosis.

Report of a Case

A white man, fifty-five years of age, came to the Mayo Clinic on April 3, 1951. He had had the usual childhood diseases. At twenty years of age he had experienced an episode of migratory polyarthritis and had remained in bed for six weeks. Subsequently he had recovered completely. In 1917, while in the United States Army, he had pneumonia with pleurisy, and ever since he had experienced cough, wheezing and expectoration of mucopurulent sputum with respiratory infections. In the fall of 1937 shortness of breath on exertion, weakness and pain in the thorax developed. The pain was located over the precordium, on the left side of the thorax and extended to the left shoulder; it was precipitated by exercise, lasted five to ten minutes and subsided quickly with rest. These symptoms produced incapacitation, and in January, 1938, he was hospitalized for six weeks. Quinidine was administered which converted an auricular fibrillation to sinus rhythm. Roentgenologic examination revealed enlargement of the heart in all diameters, left ventricular hypertrophy and calcium in the pericardium. Although a ventricular aneurysm was suspected at this time, its presence could not be established, and the radiologist felt that the possibility of an aneurysm of the left ventricle could not be definitely excluded. A diagnosis was made of chronic asthmatic bronchitis, old coronary occlusion, myocardial fibrosis, slight cardiac insufficiency, auricular fibrillation and calcified pericardium, as evidenced by the roentgenogram.

In March, 1939, the patient was readmitted to the Hines Veterans Hospital with the same findings; however, roentgenologic examination at this time revealed definite evidence of an aneurysm of the left ventricle (Fig. 1*a*). In December, 1940, he was readmitted a second time because of marked dyspnea, weakness and edema of the ankles. After three months of treatment the congestive heart failure improved; however, auricular fibrillation persisted. He was dismissed on a regimen of no extra salt diet and doses of digitalis. Subsequently, he changed his occupation from that of farming to clerking in a shoe store; he did not experience significant symptoms again until February, 1951. At this time he noted a return of dyspnea on exertion, weakness and edema of the ankles. These symptoms persisted, and in March effusion of the left pleura developed with marked dyspnea which caused him to stop work. A cardiac regimen and administrations of mercurial diuretics caused his symptoms to diminish; however, edema of the ankles persisted and his failure to get rid of this condition precipitated his visit to the clinic.

Physical Examination.—On examination it was noted that dyspnea was present when the patient was at rest. The pulse rate was 66 beats per minute and totally irregular. The blood pressure was 132 mm. of mercury systolic and 70 mm. diastolic. The patient weighed 165 pounds (74.8 kg.) and his height was 5 feet, 8 inches (172.72 cm.). The head was normal. Funduscopic examination revealed normal disks and vessels. Moist crepitant and subcrepitant rales were present throughout both lungs. The apical impulse was quite forceful and bifid in character; it was located just lateral to the anterior axillary line in the sixth intercostal space. The heart sounds were faint at the apex and absent over the base; murmurs were not present. The edge of the liver was palpable five fingerbreadths below the costal margin. Pitting edema, grade 2, of the ankles and legs was present. All arteries pulsated normally.

Results of Laboratory Tests.—The urine was alkaline and the specific gravity was 1.025; albuminuria, grade 1, was present; there was no trace of sugar, but there were 5 red blood cells per high power field. Results of the Kline test were negative; the hemoglobin content measured 12.7 gm. per 100 cc. of blood; the leukocytes numbered 9,600 per cubic millimeter, and the urea equaled 40 mg. per 100 cc. of blood. Chlorides measured 93 mEq. per liter and carbon dioxide, 25 mEq. per liter of blood. The venous pres-

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sure was 19.5 cm. of water (normal, 5 to 15 cm.), and the circulation time (ether) was 15.6 seconds. An electrocardiogram showed the following: auricular fibrillation with ventricular impulses occurring at the rate of 53 per minute with premature ventricular contractions; low amplitude QRS complex in Leads I, II and III; notched QRS complex in Leads II and III; inverted T waves in Leads I and II; iso-electric T waves in Lead III; diphasic T waves in Lead V_1; inverted T waves in Leads V_2, V_3, V_4, V_5 and V_6; depressed RS-T segment and a small Q wave in Leads V_4, V_5 and V_6. Roentgenograms of the thoracic vertebrae did not indicate any abnormality, but those of the thorax revealed evidence of thickened pleura at the base of the right lung and of the lateral wall of the thorax with calcified plaques laterally and anteriorly, fibrosis in the midportion of the right lung, and cardiac enlargement with a prominent left ventricle. Roentgenoscopically the abnormal rounded shadow involving the left contour of the heart near the apex was definitely related to the myocardial structure and exhibited a high degree of amplitude of pulsation. The lateral view showed evidence of a definite margin which appeared to be calcified. Expansile pulsations were evident as were all of the roentgenographic characteristics of a cardiac aneurysm. The previous findings of cardiac aneurysm, calcified pleura and calcified pericardium were confirmed.

While in the hospital the patient was placed on a regimen including a diet containing 0.5 gm. of sodium, rest in bed, and administrations of potassium nitrate, digitoxin (purodigin) and mercaptomerin (thiomerin). During the first four days of hospitalization he lost 7.5 pounds (3.4 kg.), became free of edema and the dyspnea improved markedly. A similar regimen was recommended to be carried out at home.

A diagnosis was made of coronary arteriosclerotic heart disease manifested by cardiac enlargement, ventricular aneurysm, chronic pericarditis (calcified), myocardial infarction (old), auricular fibrillation and congestive heart failure and of chronic bronchitis.

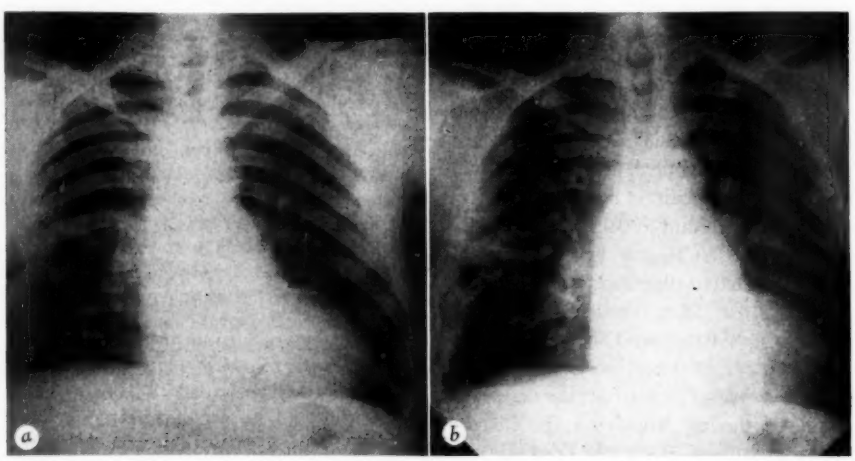

Fig. 1 (*a*) Roentgenographic evidence of a large aneurysm of the left ventricle (March, 1939; courtesy of Hines Veterans Hospital). *(b)* The same aneurysm in April, 1951, showing relatively little change in the size and configuration of the aneurysm although twelve years have elapsed since the first roentgenogram was taken. A metallic body (opposite the aneurysm) was placed on the chest wall prior to taking the roentgenogram to mark the point of maximal apical pulsation as determined by palpation.

Comment

Cardiac aneurysms occur most frequently on the anterior apical aspect of the left ventricle; that is, that portion of the myocardium supplied by the anterior descending branch of the left coronary artery. They may occur, however, on any aspect of the heart. They are usually single but may be multiple, and range in size from a tiny bulge to involvement of the entire left ventricular wall. Rowland[9] found coronary atherosclerotic heart disease, with or without hypertension, and subsequent myocardial infarction to be the etiologic agents responsible for 85 per cent of the ventricular aneurysms. These aneurysms are commoner among males than females and occur during the so-called coronary decades. Rheumatic necrosis, mycotic erosions and abscesses, syphilitic gummas, trauma and congenital defects of the myocardium have all been responsible for the formation of ventricular aneurysms. Some authors have found the incidence of ventricular aneurysm after myocardial infarction to be approximately 10 per cent,[5,6,12] and the aneurysm may develop within one week or within months after the acute episodes.[11]

Myocardial infarction is followed by extensive

necrosis of the myocardium and subsequent fibrosis with scar formation. If the patient survives, the high intraventricular pressure causes a bulging of the weakened ventricular wall during systole, and an aneurysm forms. Aneurysms develop most frequently in cases of large transmural infarcts. Advanced pericarditis usually ensues over the aneurysm.

The symptoms associated with ventricular aneurysm are those of coronary heart disease as a rule. When present, the most important physical finding is that of a separate precordial pulsation medial and superior to that of the apical impulse[2] Abnormal cardiac dullness may be present and also other signs of severe myocardial disease, such as diffuse apical impulse, poor or distant heart sounds, gallop rhythm and cardiac murmurs. In the electrocardiogram persistent elevation of the RS-T segment in the precordial leads has been associated with ventricular aneurysm,[8] and Ford and Levine[6] found this to be the most reliable electrocardiographic clue to their presence. The characteristic roentgenographic appearance of a ventricular aneurysm is that of a localized bulge in the contour of the left ventricle. Subsequent roentgenoscopic examination is usually sufficient to establish the diagnosis. Helpful criteria are myocardial angulations, incisura and abnormal pulsations, evidence of a thrombus, and pericardial adhesions.[10] Paradoxical pulsation, when present, is practically pathognomonic of ventricular aneurysm. Roentgenkymography and angiocardiography may be helpful in establishing the diagnosis. Massive ventricular aneurysms may be misinterpreted as left ventricular enlargement, and small ones may be overlooked or obscured by the presence of pulmonary congestion.

The differential diagnosis of ventricular aneurysm includes primary and metastatic tumors of the heart, lungs, pericardium and mediastinum; neurofibromas; dermoids and teratomas; aneurysms of the pulmonary artery, aorta and sinus of Valsalva; and pericardial diverticular, loculated effusions and cysts.

The prognosis in cases of ventricular aneurysm is dependent on the degree of coronary atherosclerosis present and the condition of the underlying myocardium. Death usually results from subsequent myocardial infarction, congestive heart failure, ventricular fibrillation or embolism, since rupture of an aneurysm is rare.[4]

The diagnosis of ventricular aneurysm is actually based on roentgenologic findings. This is supported further by the fact that it may be exceedingly difficult, at times, to recognize the presence of a ventricular aneurysm in specimens removed at necropsy. Perhaps we must realize that a physiologic as well as an anatomic component exists in ventricular aneurysms. Since hypertension is so frequently associated with myocardial infarction, it is an engima that the increased intraventricular pressure does not result in the formation of aneurysms more frequently. The effects of early ambulation and anticoagulation in the treatment of acute myocardial infarction and the presence of ventricular hypertrophy on formation of an aneurysm cannot be definitely evaluated at present, nor can the presence of pericarditis or an organized mural thrombus be exactly evaluated in terms of preventing rupture of the aneurysm.

Prognosis and therapy in cases of ventricular aneurysm are mandated by the severity of the patient's underlying cardiac disease and are relatively unaltered by the presence of the aneurysm; hence its diagnosis becomes largely one of academic interest. Ventricular aneurysm is actually just another manifestation of coronary heart disease.

In the case of ventricular aneurysm reported herein atypical angina pectoris (atypical location of distress but the usual exertion-rest relationship) developed in 1937. A definite history of myocardial infarction was not present; however, cardiac insufficiency forced the patient to be hospitalized in 1938. A diagnosis of coronary heart disease and myocardial fibrosis was established, but the presence of a ventricular aneurysm could not be definitely established at that time although it was strongly suspected. When the patient was readmitted to the hospital in 1939 roentgenologic examination revealed evidence of a definite aneurysm of the left ventricle. In 1941 congestive heart failure developed; however, he regained compensation and managed to lead a productive relatively unrestricted life until 1951 when decompensation again occurred. A roentgenogram of the patient's thorax taken in 1939 (Fig. 1*a*) appeared similar to one taken in 1951 (Fig. 1*b*). The large ventircular aneurysm persisted relatively unchanged in size or configuration for more than twelve years and the patient enjoyed ten years of productive, relatively asymptomatic exist-

ence during this period. His symptoms were those of coronary heart disease with congestive heart failure. Roentgenologically, the aneurysm was so large that it could have easily been misinterpreted as a left ventricular enlargement. Dermoids, teratomata and neurofibromata were excluded by roentgenograms of lateral and oblique views of the thorax and the possibility of a tumor of the myocardium or pericardium was excluded because of the duration of the enlargement and its failure to increase in size. The diagnosis was suspected at physical examination and established by cardiac roentgenoscopy. The electrocardiogram of this patient revealed evidence of severe myocardial disease and pericarditis.

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VARIETY CLUB HEART HOSPITAL REPORT

The University of Minnesota Variety Club Heart Hospital is now in full operation. Prior to October 1, 1951, only thirty-eight beds were available to patients. This was due to the acute shortage of general duty nurses. Now an additional forty beds are ready for use. Altogether, the hospital has seventy-eight beds together with an Outpatient Department, x-ray and laboratory facilities, Occupational and Recreational Therapy, and Social Service Departments. Other specialized services are available to cardiac patients in the Main University Hospitals.

Patients may be admitted to either the Outpatient Department or the Inpatient Services on the referral of their family physician. The procedure is the same as that required for admission to the Main University Hospitals.

During the first six months of operation, the cases of congenital heart disease have equaled those of acute rheumatic fever and other collagen diseases. The cases of congenital heart disease have entered the hospital for either medical or surgical treatment. The lesions most commonly treated surgically were: 1, patent ductus arteriosus; 2, coarctation of the aorta; 3, tetralogy of Fallot and 4, stenosis of the pulmonary valve. Children with rheumatic fever are treated in this hospital during the acute phase of the disease and then are discharged to their homes or to the convalescent pavilion at Glen Lake Sanatorium.

The staff is instituting research projects dealing with coronary artery disease and hypertension and consequently will be interested in seeing more of these cases than have been seen to date.

ENDOCRINOGENIC AND NEUROGENIC FACTORS IN HYPERTENSION

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DISAGREEMENT exists among investigators working upon the neurogenic origin of hypertension just as it does among those stressing renal, humoral, and endocrine origins of hypertension. All investigators agree that the central and sympathetic nervous system plays a distinct and definite part in hypertension, but the question of origin on a neurogenic or psychosomatic basis is still very debatable.

Weiss and English, Alexander, Dunbar, Binger and Ackerman and many others have shown that personality disorders are commonly associated with encephalopathies, but certain changes may occur independently of cerebral disorders.[1,4,15] Intensive investigation of the personality pattern in a few individuals suggests that hypertensives tend to display "exaggerated dependent strivings, submissiveness coupled with stubbornness, feelings of weakness and defenselessness, suppression of hostility, fear of injury and emotional detachment" that may lead to "acute emotional disorders resulting from the inefficiency of the patterns of defense against anxiety and the weakness of repressive mechanisms."[1] It has been claimed that essential hypertension may be a somatic "manifestation of a psychoneurotic condition based on excessive and inhibited hostile impulses."[16] Studies have been made by the medical departments of the Russian, English and Finnish armies during the past war which have shown that the incidence of hypertension was highest amongst front line troops, and that the incidence decreased in those farther removed from the battle zone. Russian observers noted the marked increase of severe, often deadly, but frequently reversible forms of "neurohypertension" in young individuals under war conditions. Though similar reports are not present in the American literature, there is little doubt but that this form of hypertension did exist. Hypertension has been obtained in rats by subjecting them daily to intense explosive noises; the primary cause is a sensorial stimulus with psychical response.[2a] Patients frequently assert that they are more nervous and tense following the appearance of hypertension, even when they have not been informed of the presence of an elevated blood pressure. Rest, relaxation and freedom from care have long been known to have a beneficial effect in lowering blood pressure, and for years mild sedation has been used as treatment with considerable improvement in most patients. Moreover, elimination of supratentorial activity by general anesthesia or deep narcosis frequently has a striking depressor effect. The emotional component is so variable from person to person and from time to time that it can scarcely be accorded more than an incidental role in the pathogenesis of essential hypertension. On the other hand, abnormal subcortical activity involving the medullary vasomotor centers or the autonomic nervous system may be very important and may be subject, in a measure, to influence by higher centers.

Arterial pressure is regulated by reflex pathways through pressor receptor mechanisms located principally in the carotid and aortic bodies.[2] Hering, in 1927, showed that removal of the pressor regulator system, the aortic and carotid sinus nerves, produced acute hypertension in several species of animal, especially in the rabbit. Other investigators were able to obtain chronic hypertension in rabbits lasting for several months. Koch, in 1931, obtained hypertension which lasted several months in a dog as a result of resection of the carotid sinus and of the depressor nerves. A hypertension of 250 mm. or more may accompany this operation and becomes rapidly established after the operation. Tachycardia is at times pronounced and bears a relation to the degree of hypertension. The blood pressure in this type of hypertension is usually very fluctuant, with the pressure rising to great heights under conditions of excitement, and falling to essentially constant levels when the animals are quiet and at rest. Total bilateral ganglion sympathectomy, from the cervical ganglion to the pelvic chain, will cure or prevent the hypertension produced by the section of the pressor regulator nerves.[7] The administration of xyloquinine will likewise cause a marked fall in pressure in animals with neurogenic hypertension. Extirpation of the thoracic sympathetics will cause the pulse rate to return to normal in animals with neurogenic hypertension, but the blood pressure remains elevated. Hu-

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moral factors may play a role in this type of hypertension despite the fact that it seems to be a purely nervous hypertension. The presence of adrenalin and noradrenalin in the blood and tissues of these animals has not been investigated.[2]

The correlation of the functions of the adrenal gland and of the kidney is most pertinent in the discussion of functional or neural hypertension. It is generally believed today that increased activity of the sympathetic nervous system causes general or local vasoconstriction and an increased secretion of epinephrine. This increased secretion has then been regarded as responsible for the constriction of the renal blood vessels with consequent release of renin. The comparatively recent surgical treatment of hypertension has been based on this concept. In the first operation attempted with success in 1923 only a unilateral splanchnicotomy was done, but surgical sympathectomy has gradually developed into a more and more extensive operation. The operations of Rowntree and Adam, of Peet, of Smithwick, of Craig and Adson and of Crile have become classical and are now performed with considerable frequency throughout the world. All have included denervation of the adrenal medulla and the kidney blood vessels. Results have been both encouraging and discouraging, and will not be reviewed in this paper.

Heinbecker, White, Rolf and co-workers have recently brought forth a new concept of diastolic hypertension based essentially upon the results of studies of Cushing's syndrome.[8,9,10] They have shown that circulation in Cushing's sydrome is similar in its essential characteristics to that exhibited in persons with essential hypertension. In each the skin vessels are constricted, but the degree of constriction is such that the blood flow through them under the existing increased arterial pressure is normal or only slightly reduced. In both, the efferent glomerular arterioles are constricted first, and then with progression of the disease processes the total renal flow also is reduced. It is known that in Cushing's syndrome, which may be due to a tumor of the adrenal cortex, a tumor of the ovary or thymus or to an atrophy of the hypothalamic nuclei, there is a degeneration or hyalinization of the basophil cells of the neural hypophysis; at the same time there is a relative or actual increase in number of eosinophil cells. Evidence of overaction of the eosinophil cells is indicated by a decreased insulin sensitivity, by a decrease in lymphocytes of the circulating blood, by masculinization and by development of obesity. For dogs it has been shown that eosinophils are trophic to the adrenal cortex. Evidence of lessened action of basophil cells is considered to be an elevation of blood cholesterol and a diminution of basal metabolic rate. Heinbecker and White have denervated the neural hypophysis in dogs and noted that such a procedure caused varying degrees of rise in diastolic pressure as well as results characteristic of eosinophilic overactivity and basophilic underactivity. It has been found that following the administration of an extract from the anterior lobe of the pituitary, or of adrenal cortical extract, or of simultaneous administration of both, there results a constriction of the efferent glomerular arteries. This, in turn, by decreasing the blood flow to the renal tubules, is considered to lead to a release of renin. The combined action of these three humors is held responsible for the extrarenal arteriolar narrowing and resultant diastolic hypertension. To account for the development of hypertension in persons where there are no recognizable primary causes, such as have been demonstrated in Cushing's syndrome, and who, therefore, have been designated as having essential hypertension, these workers postulate that functional influences in the central nervous system, particularly in the frontal lobes of the cerebral cortex, normally may depress the hypothalamic nuclei which control the secretion of the neural hypophysis. The anatomic and physiologic bases for such influences are well established. On depression of the functional activity of these nuclei there results a diminution in the secretion of the neural hypophysis. This, on the basis of experimental evidence presented, can be assumed to result in a stimulation of the eosinophil cells of the hypophysis and these in turn stimulate the adrenal cortex to greater activity. The combined influence of the anterior pituitary hormone and of the adrenal cortical hormone is to effect a constriction of the efferent glomerular arterioles of the kidney and a release of renin. The combined action of the three humors here, as in Cushing's syndrome, is held responsible for the arteriolar narrowing and the development of diastolic hypertension.

The existence of human hypertension of purely endocrine origin is a generally accepted fact; the hypertensive state associated with pheochromocytoma of the adrenal is perhaps the best known and understood example of this type of

hypertension.[3,6] The adrenal gland itself has been shown to play the largest role of the endocrine glands in the development of essential hypertension even if it has been accorded a secondary role by some investigators. Complete excision of both adrenals in the dog interferes with the development of hypertension due to constriction of the main renal arteries unless supportive and substitution therapy is given. In hypertensive animals also, hypophysectomy causes a fall in the blood pressure which is due to adrenal hypofunction; adrenocorticotrophin restores the pressure to the former hypertensive levels.[11] The removal of the posterior lobe of the hypophysis has no action on the blood pressure of normal mammals or animals with renal hypertension. When only a small part of the cortex of a single adrenal remains, there is no interference with the development of experimental hypertension in the dog. If both adrenals of a hypertensive animal are removed, the blood pressure promptly drops to normal.

Great attention has been focused upon the work of Selye within the past three or four years in regard to the subject of hormonal factors in the production of hypertension.[13,14] This investigator, the originator of the general-adaptation-syndrome, believes that stress (or "disease of adaptation," the results of excessive or abnormal adaptive reactions to stress) initiates a chain reaction which ultimately causes an increased blood pressure. The stress, through unidentified pathways, possibly blood-borne tissue catabolites, sets into action a hypophyseo-adrenal defense mechanism. It first acts upon the hypophysis, causing it to increase corticotrophin production at the expense of a decreased production of gonadotropins, luteotrophin, growth hormone, et cetera. The rise in corticotrophic hormone secretion causes enlargement of the adrenal cortex with signs of increased corticoid production. These corticoids in turn elicit changes in organic (such as gluco-corticoids producing increased glyconeogenesis) and inorganic (mineralo-corticoids) metabolism as well as such changes as atrophy of the thymus and other lymphatic tissues, changes in the joints, periarteritis nodosa, myocarditis and nephrosclerosis. The exact mechanism of action of the mineral-corticoids upon the kidney (nephrosclerosis) is unknown but probably is dependent upon their action on electrolyte metabolism and sodium retention; the withdrawal of sodium inhibits their effect and purified gluco-corticoids cause no nephrosclerosis. The nephrosclerotic kidney then presumably produces an increased amount of renin and the hypertensive state ensues.

The removal of the anterior lobe of the hypophysis produces a fall in the blood pressure in normal dogs and rats. In the latter, the normal level of the blood pressure can be restored in part by the administration of desoxycorticosterone, totally by the administration of adrenocorticotrophine. The blood pressure is restored in these animals by the administration of desoxycorticosterone or of sodium salts and in the rat by the administration of cortisone. In normal man cortisone has no effect on the blood pressure, but it does cause an increase in pressure in patients with Addison's disease.[12]

Conclusions

The etiology of hypertension is unknown. Some of the psychogenic, neurogenic and endocrinogenic factors which play a part in its formation have been discussed.

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President's Letter

RETROSPECTIVELY SPEAKING

Looking back over the present year, one feels that the medical profession, state and national, has accomplished much—scientifically, through physicians' constant endeavors to be better prepared for their work; educationally, in presenting the position and ideals of medicine to the public, in their context of American life and thought; economically and politically, through watchful caution and co-ordinated action.

While we are, happily, not so near socialized medicine and national socialism as we were in 1950, the fight is not won. A system of "creeping socialism" is being urged on us, a bit here and more there, with baits like free hospitalization for those over 65. Do not think that the groups fostering these plans have any thought of relaxing. When we see the evidence of bureaucracy at work in our national government, we begin to realize what a rich hunting ground socialized medicine would provide.

The heavy hand of bureaucracy has shown itself in many ways: its punitive action may be interpreted from an example in Indiana, where the federal security administrator cut off $18,000,000 in relief money because the state was releasing the names of relief recipients; its economic hold on the states may be illustrated by the way tax money is trimmed by federal costs of administration before being sent back to the states—and that with restrictions as to how it may be spent.

I know that the profession is alert to all the aspects of the dangerously expandable area known as bureaucracy, and in the year ahead will move forward courageously and effectively to combat any threatening ideologies.

Now, at this time, I would like to thank the many members of the profession who were kind enough to advise and help me and make the past year a rich experience for me. I have been amazed to note how much hard work is being done by the officers in various communities, the House of Delegates and the Council. I have been greatly pleased to note what appears to be an increasing interest in the affairs of the medical society, and especially by younger groups. This is as it should be.

Allow me here to extend to each one of you my best wishes for the Christmas season and a New Year that will be a happy one for you.

J. F. Norman

President, Minnesota State Medical Association

Editorial

CARL B. DRAKE, M.D., *Editor;* GEORGE EARL, M.D., HENRY L. ULRICH, M.D., *Associate Editors*

NEW VA LAW

RECENT legislation provides that "service connection may be presumed" for all psychosis cases developing within two years after discharge from service for World War II veterans and those in service since the start of the Korean War. It is estimated that this law may require the Veterans Administration to provide for some 9,000 veterans who before passage of the law were listed as suffering from non-service-connected disability and entitled to hospitalization but not out-patient care, only if facilities were available.

All of the 53,000 neuropsychiatric beds of the VA hospitals are at present occupied by psychiatric patients, some with non-service-connected disabilities. These cannot very well be discharged. In order to comply with this new law, these 9,000 veterans now entitled to hospital care will have to be admitted to the hospitals in preference to those with non-service-connected disabilities who may be admitted only if there is room. An undetermined number of these 9,000 psychiatric veterans are now in state institutions and presumably may continue there at VA expense. How many more veterans will eventually be added to this category is impossible to estimate.

IMPROVING PUBLIC RELATIONS

GOOD public relations depends on mutual confidence and respect between the individual patient and physician. Disruption of this relationship is in most cases due to misunderstanding in regard to money matters. This has been demonstrated by the grievance committees established by medical societies all over the country, which enable dissatisfied patients to air their complaints regarding their medical care. In most cases, it has been found, complaints of overcharging by physicians have been cleared up by frank discussions between patient and physician.

With the idea of preventing these misunderstandings a plaque for placing in the physician's office has been prepared* at a nominal charge which simply and straightforwardly states.

TO ALL MY PATIENTS

I invite you to discuss frankly with me any question regarding my services or my fees. The best medical service is based on a friendly mutual understanding between doctor and patient.

The physician who places this plaque in his office indicates his willingness to discuss all matters regarding his services including fees, either before or after they are rendered. This does not mean that he wishes to encourage his patients to shop around with the idea of choosing the cheapest service available. Medical service should involve more than the mere exchange of dollars. Extenuating circumstances such as financial status should be and are taken into consideration by physicians.

We believe that the placing of this plaque in a physician's office is a step in the right direction towards improving public relations.

*This plaque, suitable for hanging in a physician's office, may be obtained from AMA headquarters for one dollar.

TAXES AND OUR CHANGING AGE DISTRIBUTION

WE OF the medical profession are inclined to point with pride to the increase in life expectancy from forty-nine in 1900 to sixty-nine in 1950. The possibility and even likelihood of serious economic repercussions from this improvement in life expectancy are brought out by Frank G. Dickinson, director of the Bureau of Medical Economic Reserach of the AMA, in a recent address.†

Whereas, the population of the United States has doubled since 1900, the number of people in the country over sixty-five has quadrupled. By 1980, one out of every eight in the country's population will be over sixty-five. Two of the eight will be under the age of twenty. This

†Reprint from Problems of America's Aging Population, Vol. I, Institute of Gerentology Series, University of Florida Press, Gainesville, 1951.

means that five of each group of eight in the population will be in the wage-earning age and will have to support three others—two under the age of twenty and one over sixty-five.

One may be inclined to argue that Social Security payments will largely take care of the support of those over sixty-five. When we consider that the funds contributed by wage earner and employer under Social Security consist largely of merely stacks of non-negotiable but interest-bearing Government bonds in the Treasury Department and that when claims exceed current contributions they will be paid out of general tax revenues, the seriousness of the future tax situation becomes apparent.

As Dickinson points out, the group of the indigent aged is going to elicit sympathy in the future as the most needy, economically speaking. At the time of Karl Marx and his contemporaries, it was the laborer and factory worker who needed sympathy and had to fight for enough food and clothing in order to survive. Today, this group is not in need of these essentials and is, indeed, well supplied with the comforts of life. Any increase in wages for this group will more likely be spent on increased comforts of living, such as automobiles, electric refrigerators, televisions, et cetera.

Whereas, in 1900, 24 per cent of the population was over fifty years of age, this percentage had increased to 33 per cent in 1947. This constitutes a large proportion of the voters. What if some demagogue should come along advocating free hospital care, free medical care, free housing, free food for those over sixty-five. Free hospital care for those over sixty-five and covered by Social Security has already been advocated by Oscar Ewing. The ever-increasing proportion of voters in the older age group will present an ever-increasing danger of legislation favoring this group at the expense of the productive group. This would logically result in co-operation between laborer and employer in the political field to prevent taxes from becoming unbearable and a shifting of conflict from the field of employer and laborer to that of youth and old age.

Townsendism may not have been a serious issue in years past. According to Dickinson, it may prove as important an issue in the next fifty years as the doctrines of Karl Marx were in the last half century.

Dickinson warns (and he is right) that we are creating a world in which our children will have little to look forward to but a life of work, earning and paying taxes.

HOW DOES ONE OBTAIN A POSTMORTEM EXAMINATION?

IN the so-called private hospital, the method of obtaining a postmortem examination is antiquated and decidedly cumbersome. The teaching hospitals have a high index of psychological pressure and a much larger staff which affords them higher ratios of examinations. Even in these hospitals the method could be improved.

The difficulty in the private hospital is due to a combination of reasons. There is the persistence of the old idea that a patient in the hospital is the property of the clinician. Again, only recently have hospitals developed facilities for well-documented records. When compared with other functions of hospital management, the official part of the business of getting a postmortem examination has not been developed.

Morally and legally, when a patient enters a hospital the patient is a charge of the hospital. The clinician who attends the patient is merely the therapeutic agent. The time has arrived when the management should assume the responsibility of obtaining necropsy records. This record is a hospital record. Every possible opportunity has developed to perform postmortems, but the method of obtaining the examination by the management has been neglected. The management still defers to the clinician's wishes and leaves the onus of getting the postmortem examination entirely to the clinician. There lies the crux of the cause for low ratios in our hospitals.

Let us detail the present method. With variations, this is the usual mode of procedure.* When a clinician is notified that a death has occurred, he urges the intern to "get a post." If the intern fails he, himself, tries to "get a post." If he has had success, he must notify the pathologist; he must arrange the time at the mortuary; he must obtain the writ of consent; he must present it to the pathologist. All of this is time-consuming and a heavy burden on the clinician. All of the above duties could and should be a part of the official business of the hospital.

*The description is based on experience at Abbott, Asbury, Northwestern, Deaconess, Eitel and Swedish Hospitals, of Minneapolis.

Think of what a pressure or weight of persuasion can be put on the relatives if the approach came originally from the hospital. It would have an authoritative note which the clinician sadly lacks in this trying period.

The following plan is suggested in which the hospital takes its proper place to obtain a necropsy report:

1. *The nursing staff:* As soon as a death occurs, the head nurse in charge should suggest to the relatives, "Of course, you intend to have a postmortem examination."

2. *The house staff:* The house officer or intern should immediately acquaint the relatives of the desirability of postmortem examination; and request it, with the permit all ready to sign.

3. *The hospital office:* The "front" official in charge at the time should urge the importance of an autopsy, with a permit ready to be signed.

These three stages might be called the bombardment or attack on the relatives.

Meanwhile, the pathologist should have been alerted concerning the death. He should also appear as an official of the hospital to lend his persuasion for the examination. If all these approaches fail, the clinician should and would make the final appeal.

This approach, with its impersonal quality, will emphasize the importance of postmortem examinations in the public mind. Furthermore, the hospital will have taken its proper place in improving its scientific status—and assumed the responsibility in obtaining the records for its archives.

To reiterate, "getting a post" is an administrative job and not the clinician's. The latter's attitude should be merely one of wanting a postmortem examination and always being willing to assist in getting one—that is, when all official efforts have failed.

—An Internist.

The above editorial was submitted to a hospital administrator and brought the following response:

After really thoughtful study of your paper, "How Does One Obtain a Postmortem Examination," I find that:

First: Your proposal jars many of my preconceived ideas of physician-patient (family) relationships.

Secondly: Under your arrangement I, unfortunately, am of the opinion that the number of staff men, interns and residents attending an autopsy on their patient would be less than at the present very low ebb.

Third: While to secure postmortem examinations by hospital authorities and thus keep up or increase the autopsy rate is meritorious, I have the feeling that this is too great a shift of interest in medical science from the physician to the corporation.

Fourth: From a public relations point of view, morticians are pretty universally trying to discredit hospitals where higher autopsy rates prevail, unless the rapport with the physicians is good.

Fifth: You are asking the hospital to keep the progress of medicine in motion.

Sixth: It may seem an extreme point of view, but when the hospital steps in at death, would an attitude grow up that it was a face-saving procedure because the physician had a failure?

Seventh: As a layman with a genuine interest in medical education, I have always assumed that the autopsy in the presence of the attending and house staff was the final opportunity for precise study of diseases in patients dying under hospital care, and occasionally to determine whether the hospital had been negligent in its care. I believe that the autopsy should, for best use, remain the objective concern of medicine.

Lastly, the body in life is, I believe, the resting place of one's immortal soul. Perhaps for this reason, rational or otherwise, Christ is called the great physician. Although in death the soul departs, the humble privilege of further exploration in the field of God's great handiwork should remain with his disciple, the physician.

Sincerely,

Administrator.

MEDICAL SOCIETY DUES

Now is the time for billing and paying medical society dues. The total for county, state and American Medical Association dues may seem a considerable item, but when one adjusts for a 54-cent dollar it doesn't seem so steep. Of course, when the bill includes unpaid AMA dues for 1950, the total is $25.00 greater than it would otherwise be, for the Council of the Minnesota State Medical Association voted at its meeting September 23, 1951, that county societies should continue billing unpaid AMA dues for 1950 and 1951 until paid.

The declaration of an assessment by the AMA in 1949 for financing the educational campaign to ward off government control of medical practice, which was voluntary and did not affect one's medical society membership, laid the groundwork for confusion. In 1950 the House of Delegates voted dues of $25.00 for the year, nonpayment of which sacrificed membership in the AMA but not in county society or state associations. In 1950 the AMA dues did not include subscription to *The Journal AMA* which was $12.00 additional. In 1951 the AMA dues of $25.00 included a subscription to *The Journal AMA*, separate subscription to which is now $15.00. This increase in the subscription price to $15.00 was made necessary by the increase in printing costs which has been considerable in the past decade.

Confusion was further enhanced when those who subscribed to *The Journal AMA* in 1950 continued to receive it in 1951, even if they did not pay their AMA dues for 1951. On the other hand, those who paid their AMA dues for 1951 but had failed to pay their 1950 dues did not receive *The Journal AMA*.

Further confusion has been caused by the existence of a special membership known as a "fellow." In 1951 this cost an extra $5.00. Beginning with the year 1952 there will be just one membership and no more "fellows."

The simple solution for each member in arrears would be to pay up in full, shoulder his share of the AMA expenses and take his place with the nearly 150,000 other physicians in the country who are banded together for the advancement of the science and art of medicine and the betterment of public health.

For $15.00 of his $25.00 AMA dues, the member receives $15.00 worth of *The Journal AMA*—one large number a week—or he may substitute a subscription to any one of the specialty journals. The balance of the $25.00—or $10.00—helps support the multitude of activities of the AMA. The only other income the AMA has is from advertisements in *The Journal AMA* and the nine specialty journals published by our national organization. Some of these journals are not self-supporting.

When we consider what we receive in return for our various medical society dues, it becomes evident that we receive more than our money's worth.

THE CHANGING MOLE

(Continued from Page 1158)

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GREATER UNIVERSITY FUND

In 1950, more than 2,300 alumni and friends of the University of Minnesota contributed over $100,000 to the many projects supported through the Greater University Fund.

Some of the activities made possible by the Fund are: A thermocouple that measures the rate of chicken-freezing in Poultry Husbandry; a tele-binocular used to improve children's reading in the Psycho-Educational Clinic; an electronic computor for nerve study in Physics; a machine with which Veterinary Medicine will record the heart and respiratory sounds of animals.

Not all of the Fund goes to scientific research. Each year at least forty freshmen are awarded $250.00 in Minnesota Alumni Scholarships. Three outstanding graduate students this year will receive Fellowships, each amounting to $1,200, plus tuition.

The Greater University Fund is sponsored by the Minnesota Alumni Association and is now conducting its fourth annual drive with $125,000 the goal.

Here is an opportunity to assist in a worthwhile project. Contributions which are income-tax deductable may be sent to the Greater University Fund, Coffman Memorial Union, University of Minnesota, Minneapolis 14, Minnesota.

A BOTANICAL CONSIDERATION OF THE WEED OLEORESIN PROBLEM

(Continued from Page 1173)

3. Not only are there differences in sensitizing capacity between botanical tribes but also there are differences in sensitizing capacity within a given tribe.

4. The probably antigenic relationships are indicated with respect to tribes *Anthemideae* (dog fennel) and *Heliantheae* (sunflower).

5. The profile of our cases dealing with the sunflower tribe *(Heliantheae)* resembles that of Shelmire's study of this same group of weeds.

6. Biologic studies will be carried out to determine further allergenic relationships between the various oleoresinous materials.

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869 Lowry Medical Arts Building

Medical Economics

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
George Earl, M.D., Chairman

"FIGHT FIRE WITH FIRE" IOWA DOCTOR URGES

Members of the North Central Medical Conference, November 11, heard Dr. Fred Sternagel, West Des Moines, Iowa, warn that it is still necessary to be on guard against medicine's enemies and fight fire with fire. In his presidential address, Dr. Sternagel said, "We must be realists and fight fire with fire. We must meet every criticism of our profession, regardless of the source from which it may spring. We must have a definite and positive program of action."

Reviewing the medical profession's fight against socialized medicine in America, Dr. Sternagel stated:

"The battle lines have been drawn. Those who are pushing this country into Socialism are on one side. On the other side there are many staunch defenders of traditional American freedom, but the immediate contest of the moment is between the leaders of the welfare state theory and American physicians. . . . We have been drawn into a fight, so let us be good fighters. We can't win this fight by name-calling or deploring the changes of the times. If we are going to save freedom we must do so by methods of freedom. We must prove that freedom is best for most people."

Pendulum Swinging Back

"We are firmly convinced," said Dr. Sternagel, "that the time has arrived for the pendulum to start swinging in the other direction. . . . We have had our backs to the wall, but we are gaining strength to surge forward and make our position secure. It may be part of the world-wide reaction against Socialism in England and imperialistic Communism."

Dr. Sternagel felt that this reaction does not mean that people no longer want the good things in life. He said:

". . . they are not convinced they can obtain such blessings through Socialism and Communism. They want to give free enterprise one more chance. This may be our last chance! If we fail to take advantage of it, what we think now is a reaction to the welfare state in our own government and Socialism and Communism abroad may be merely a pause—the quiet before the storm—the gathering of forces for a new onrush which we cannot stop.

"The medical profession is being weighed in the balance. Let us not be found wanting! This is our fight for freedom! Let's win that fight!"

HOSPITAL CONSTRUCTION PROGRESS REPORTED

A total of thirty-nine projects in Minnesota are now receiving aid under the Hospital Survey and Construction Program, with a total of 477 beds, according to a report presented by A. J. Chesley, M.D., Secretary and Executive Officer, Minnesota State Department of Health. He states:

"In addition to the thirty general hospitals, nine other projects are receiving assistance under the Hospital Survey and Construction Program as follows: three chronic disease units with a total of 287 beds; three mental disease hospitals (includes a receiving unit of a State mental hospital and two psychiatric units of general hospitals) plus a psychiatric unit in one of the general hospital additions shown above, total 190 beds; two public health centers, plus 180 beds in two nurses' homes, one of which is included as a part of one of the general hospital projects listed above. (Total 477 beds, exclusive of beds in nurses' homes.)"

Fifty General Hospitals Built

The report also shows that a considerable number of hospitals have been constructed without the aid of the federal grants. General hospitals completed during the past fifteen years include a total of fifty, with an aggregate of 1,928 beds. In addition to these, the report reveals that a 122-bed chronic disease unit was opened; the State of Minnesota is providing mental disease facilities at six of the State hospitals as follows: eight geriatric units with a total of 1,200 beds; three receiving units with a total of 198 beds; a continued treatment building with 204 beds and a 112-bed medical-surgical unit. Also a 251-bed tuberculosis unit was completed; a thirty-seven-bed neuro-

logical rehabilitation unit was added to another hospital; and seventy-one additional beds for the care of tuberculosis patients were added to another hospital. This makes a total of 2,195 beds added to the state's hospital facilities without the assistance of federal funds.

Goal: A Co-ordinated System

An over-all goal for the hospital construction program is presented by this report:

"As the construction of hospitals progresses, it is hoped that institutions and their services will develop toward a co-ordinated system which can provide complete care for all patients. The existing and proposed hospitals are classified as base, regional, area and rural hospitals. . . ."

Commends Co-operating Groups

The report lauds the efforts of co-operating organizations to bring about certain improvements in existing services in Minnesota: "Efforts are being directed toward the development of better services in hospitals. This includes an expansion of existing training programs for hospital personnel as well as a general educational program relative to the need for and the use of improved services, particularly in the more rural areas of Minnesota. . . . It is the intent of these and other interested organizations to develop plans for meeting deficiencies in these services in Minnesota."

FOES OF HOSPITALIZATION FOR AGED CALLED "HEARTLESS"

Oscar Ewing, in his continued attempts to get his schemes approved for federal application, is using mass psychology to create sympathy for the recipients of one of his welfare plans. In announcing the plan to provide free hospitalization for people over sixty-five, which would come out of Social Security funds, he did not believe that there would be much opposition to his plan. "I can't conceive that anyone with a heart would oppose this. This is something they would have paid for themselves in insurance. . . . I can't believe doctors don't want sick people to have attention."

Thus, Ewing challenges doctors to be heartless and deny sick old people the right to a free handout from the government. Ewing's play for sympathy should be studied for what it really is—an attempt to gain support for one of his pet programs with welfare state leanings.

It is because the medical profession has a deep concern for sick people, no matter what age, that it has taken up the fight to keep the nation free from such socialistic plans. Doctors are not denying sick people the chance for care, but they are constantly striving for improvements which will make it easier to provide more and better care. Doctors, and other forward-looking Americans, might stop and consider that, if they "have a heart" in this case, they may well pay for it too strongly through further taxes and many more government controls, contingent upon this and other similar plans. Mr. Ewing may well take the advice of Mr. John Wanamaker: "No man starting out need ever go over the road in the dark, if he take with him the light of other men's experience." Oscar Ewing should look to England for his inspiration, taking heed of that "light of other men's experience."

MAGAZINE MAY EVEN "TIP HAT" TO EWING

While one medical journal advises doctors to "take the incident as indicative of their urgent responsibility to 'wax heartless' again before it's too late," the October issue of *Medical Economics* says that "the new Ewing proposal might well be the nudge we've needed."

"The day may yet come when we will quietly tip our hats in the direction of Oscar Ewing. By proposing 'free' hospital care for Social Security beneficiaries, he has focused attention on a genuine trouble spot—one that medical, hospital, and insurance men have side-stepped for too long."

Discussing this fundamental problem of the older age group, the publication feels that a basic need is the building up of old people's purchasing power. Under the Social Security Act, pension recipients get an average of $48 a month—far below an amount enabling economic freedom. The magazine states that Ewing's proposal "would, after all, merely superimpose a towering hospitalization giveaway on an unstable Social Security base."

MEDICINE URGED TO TAKE LEAD

In an odd sort of way, this whole problem of the aging population is the fault, or result of, the

progress of medical science. Every day almost 3,000 Americans reach the age of sixty-five. On the average, they have thirteen years of retirement ahead of them. The astonishing increase in our sixty-five plus population stems in large part from medical advances. Thus, the magazine asserts, "it's logical that we take the lead in coping with the oldsters' health-cost plight."

SOFT SECURITY "NOT WORTH IT"

A recent editorial in the *New England Journal of Medicine* expresses vividly the thoughts of many who are weary of hearing about the blessings of security, the payment for which comes out of earnings, regardless of ability to pay. Classing the rush for security as a purchase too full of risks, and not worth the inert status security represents, the *Journal* states:

"In the midst of unrest and danger and threats to the cherished social and economic institutions of the world, it is natural that there should be a turning toward whatever seems to offer security—or whatever can be labeled by that name. Security has been the goal of the common man since first he blockaded himself in his cave; it has been the intermittent, always vulnerable achievement of possessive man since flocks first were grazed in the Fertile Crescent between the twin rivers of Mesopotamia.

The *Journal* urges Americans to have the courage to say "no" to what appears to be Utopian, for it contains within it, the power of its own, and the individual's, destruction.

"The purchase of a soft security, however, is always made at too great a price for the inert thing that it represents. A life untroubled by want from the cradle to the grave would be also a life unmarked by glorious hours, untouched by lofty emotions and unstirred by noble risks; it would be no more than a vicarious participation in the tumultuous affairs of the world—and the only guarantee that might accompany it could concern only its inevitable ending.

"The only answer to the challenge appears to lie not in a passive apparent security—a withdrawal from the realities of existence purchased with inflated currency—but in the reactivation of certain basic virtues that are dependent on the courage to face facts as they are. 'The courage is needed to say "No" when to say "yes" is easier and pleasanter; to recognize the truth and to admit the inability to find the truth.' Courage is required to remain humble when others are vaunting themselves; to continue a righteous struggle when the effort seems doomed to failure; to fail, still striving for what has been accepted as the right."

UNRESOLVED PNEUMONIA

(Continued from Page 1168)

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Despite the knowledge that Federal finances are in a critical phase, the Administration, while paying lip service to economy, contends that the Federal budget is tight, and offers stubborn resistance toward any retrenchment. In the meantime, Federal expenditures are rolling along in snowball fashion, with current outlays at the annual rate of $71.6 billion. The addition of state and local expenditures brings total governmental costs for the present fiscal year to over $92 billion. This sum is equivalent to $2,315 per family, or nearly four and one-half times the burden per family in 1939.

The time has come when the American people must take a vigorous and courageous stand for national solvency and be mindful of the warnings issued by Thomas Jefferson more than a century ago when he said: "We must make our selection between economy and liberty, or profusion and servitude." Congressmen who are fighting for economy in government should be given wholehearted support.

Everyone concerned about the welfare of the country should join the nation-wide movement to demand action on the Hoover Commission's recommendations for the elimination of waste in the Federal Government. To be victorious, this group must match the zeal and determination of the spenders who are squandering our substance and undermining the foundations of our freedom.—*Spotlight* No. 83. Published by committee for Constitutional Government, Inc.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

230 Lowry Medical Arts Bldg., Saint Paul, Minnesota

Julian F. Du Bois, M.D., Secretary

PHYSICIANS LICENSED FEBRUARY 16, 1951

January 1951 Examination

Name	*School*		*Address*
BARRETT, Jack William	St. Louis U.	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.
BEATON, Joseph Gordon	U. of Minnesota	MB 1942 MD 1943	101½ N. Minnesota St., New Ulm, Minn.
COWLEY, Howard Stephen	Creighton U.	MD 1941	102 110 2nd Ave. S.W., Rochester, Minn.
DOBSON, Ross Allan	U. of Toronto	MD 1949	102 110 2nd Ave. S.W., Rochester, Minn.
DOERR, John Charles	U. of Louisville	MD 1944	102 110 2nd Ave. S.W., Rochester, Minn.
GULLEY, John Lewis	U. of Toronto	MD 1945	102 110 2nd Ave. S.W., Rochester, Minn.
KELLY, John Moyer	U. of West. Ont. Can.	MD 1945	511 Lowry Med. Arts Bldg., St. Paul, Minn.
MAXWELL, John Burchiel	U. of Louisville	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.
McMANUS, Terence Burton	U. of Minnesota	MB 1950	Bank of Commerce Bldg., Milbank, S. D.
MEULI, Earle Joseph	U. of Wisconsin	MD 1949	Maunaloa, Molokai, T. H.
MICHAEL, Isaac Eldrew	U. of Chicago Rush Med. College	MD 1942	102 110 2nd Ave. S.W., Rochester, Minn.
MICHALEK, Louis Frank	U. of Washington	MD 1950	U. of S. D. Med. School, Vermilion, S. D.
MILLIKAN, Clark Harold	U. of Kansas	MD 1939	102 110 2nd Ave. S.W., Rochester, Minn.
MORRIS, Kenneth Lyle	Northwestern U.	MB 1942 MD 1943	102 110 2nd Ave. S.W., Rochester, Minn.
NEARY, Angus James	Dalhousie U., Can.	MD 1948	102 110 2nd Ave. S.W., Rochester, Minn.
REESE, Gilbert Alan	U. of Wisconsin	MD 1949	102 110 2nd Ave. S.W., Rochester, Minn.
SATHER, Howard L.	Marquette U.	MD 1948	St. Luke's Hospital, Chicago, Ill.
SCHERMAN, Quinten	U. of Oregon	MD 1945	102 110 2nd Ave. S.W., Rochester, Minn.
THOMPSON, Jr., Ollie Hammond	Johns Hopkins U.	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.
WHEELER, Gilbert Stephen	U. of Manitoba	MD 1949	Roseau, Minn.
WOOD, Earl Howard	U. of Minnesota	MB 1941 MD 1941	102 110 2nd Ave. S.W., Rochester, Minn.

Reciprocity Candidates

Name	*School*		*Address*
BARRY, Jr., Maurice J.	U. of Indiana	MD 1944	102 110 2nd Ave. S.W., Rochester, Minn.
BENSON, Alfred Homer	Loyola U.	MD 1940	105½ 1st St. S.E., Little Falls Minn.
BRENNAN, James W.	U. of Buffalo	MD 1938	570 Delaware Ave., Buffalo, N. Y.
BUCHANAN, Gerald S.	Coll. of Med. Evang.	MD 1950	Deer River Hospital Clinic, Deer River, Minn.
ELLERTON, Jr., Leonard Melvin	U. of Iowa	MD 1946	126 E. William St., Albert Lea, Minn.
GAUNT, William Danforth	Washington U. St. Louis, Mo.	MD 1948	102 110 2nd Ave. S.W., Rochester, Minn.
HELDEN, Rudolph Andrew	Jefferson Med. Col.	MD 1945	102 110 2nd Ave. S.W., Rochester, Minn.
MANNING, Phil Richard	U. So. Calif.	MD 1948	102 110 2nd Ave. S.W., Rochester, Minn.
MARKS, Richard Lewis	Vanderbilt U.	MD 1936	137 Vine Ave., Park Ridge, Ill.
MARTIN, Harold R.	U. of Indiana	MD 1944	102 110 2nd Ave. S.W., Rochester, Minn.
McFARLANE, John Alvin	Washington U. St. Louis, Mo.	MD 1948	102 110 2nd Ave. S.W., Rochester, Minn.
MORTENSEN, J. D.	U. of So. Calif.	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.
MULDER, Donald William	Marquette U.	MD 1943	102 110 2nd Ave. S.W., Rochester, Minn.
RUDESILL, Robert Louis	U. of Indiana	MD 1947	102 110 2nd Ave. S.W., Rochester, Minn.
SCHLEMENSON, Saul Melvin	U. of Michigan	MD 1943	410 Doctors' Bldg., Minneapolis, Minn.
SHAFFER, James Milton	Western Reserve U.	MD 1946	U. of Minn., Dept. Pharm., Minneapolis, Minn.
SIMON, Thomas Raymond	St. Louis U.	MD 1945	St. Cloud Hospital, St. Cloud, Minn.
SMITH, CHARLES Felps	U. of Indiana	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.
SPENCER, Jr., Fred Davis	U. of Texas	MD 1942	102 110 2nd Ave. S.W., Rochester, Minn.
STEINHILBER, Richard Miller	Hahnemann Med. Col.	MD 1945	102 110 2nd Ave. S.W., Rochester, Minn.
WAGONER, James Marion	U. of Indiana	MD 1945	Harmony, Minn.
WAUD, Robert Edgar	St. Louis U.	MD 1947	AUS: E. 115 S. 4th St., Grangeville, Ida.
WUEST, Jr., John Henry	U. of Louisville	MD 1947	102 110 2nd Ave. S.W., Rochester, Minn.

National Board Candidates

Name	*School*		*Address*
ANDERSON, Harris Renard	Northwestern U.	MB 1949 MD 1950	3882 Sch. Squadron, S. A. M.. Box 59, Gunter A.F.B., Montgomery, Ala.
BAUER, Donald deForest	McGill U.	MD 1942	Miller Hospital, St. Paul, Minn.
EDWARDS, Laura Elaine	U. of Minnesota	MB 1948 MD 1949	c/o Baptist Gen. Conference of America, 912 Belmont Ave., Chicago, Ill.

FAHL, James Cox	Harvard U.	MD 1948	102 110 2nd Ave. S.W., Rochester, Minn.
GUNDERSEN, Gunnar Adolf	Harvard U.	MD 1948	Ebner's Coulee, La Crosse, Wis.
HERBST, William Parker	Harvard U.	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.
HUBBARD, Jr., Thomas Brannon	Columbia U.	MD 1942	U. of M. Hospitals, Minneapolis, Minn.
KINGSLEY, Buell Chapin	U. of Oregon	MD 1948	U. S. Naval Hospital, Bremerton Wash.
KRUMBACH, Ronald William	Syracuse U.	MD 1948	U. of M. Hospitals, Minneapolis, Minn.
LOWRY, Jeanette Kraemer	U. of Minnesota	MB 1948 MD 1949	U. of M. Hospitals, Minneapolis, Minn.
LUBIN, Robert Irving	U. of Iowa	MD 1949	Vet. Adm. Hospital, Minneapolis, Minn.
LYNN, Thomas Edward	Northwestern U.	MB 1949 MD 1950	102 110 2nd Ave. S.W., Rochester, Minn.
OGBURN, Paul Lanier	Duke U.	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.
PERSON, Waldemar Norman	Loyola U.	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.
PHILLIPS, John Wesley	St. Louis U.	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.
PIERCE, Jack Reed	U. of Buffalo	MD 1943	401 5 So. 1st St., Virginia, Minn.
SMITH, Meredith Parks	U. of Maryland	MD 1949	102 110 2nd Ave. S.W., Rochester, Minn.

PHYSICIANS LICENSED MAY 18, 1951

April 1951 Examination

Name	*School*		*Address*
BERNIER, Merrill Joseph	Northwestern U.	MB 1949 MD 1950	1234 Division St., No. St. Paul, Minn.
CRAYCHEE, Walter Albert	Temple U.	MD 1945	118 S. Main, Fairmont, Minn.
DONIN, Jerry Frank	U. of Manitoba	MD 1950	102 110 2nd Ave. S.W., Rochester, Minn.
DORNBACH, Robert Arthur	Georgetown U.	MD 1950	Mpls. Gen. Hospital, Minneapolis, Minn.
FIELD, Harold C.	Northwestern U.	MB 1950 MD 1951	106 S. 4th St., Guthrie Center, Iowa
FLORA, George Claude	Temple U.	MD 1950	St. Barnabas Hosp., Minneapolis, Minn.
KRAMER, Stephen Elliott	Georgetown U.	MD 1933	Anoka State Hospital, Anoka Minn.
LAMB, Henry Douglas	Guy's Hosp. Sch., LMSSA U. of London, England	1937	Students Health Serv., U. of Minnesota, Minneapolis, Minn.
LOCKHART, Henry Bent	U. of Toronto	MD 1944	102 110 2nd Ave. S.W., Rochester, Minn.
LORENTZEN, Ernest Stanley	Northwestern U.	MB 1950	Detroit Lakes, Minn.
McKINLAY, Gordon Lynn	U. of Chicago	MD 1950	Mpls. Gen. Hospital, Minneapolis, Minn.
MICHELA, Bernard Joseph	Cornell U.	MD 1949	St. Mary's Hospital, Duluth, Minn.
MICHELSON, Therese	Creighton U.	MD 1949	3517 Oakland Ave., Minneapolis, Minn.
QUIGGLE, Arthur Benajhar	U. of Minnesota	MB 1950	U. of M. Hospitals, Minneapolis, Minn.
RUFF, John Douglas	Med. Col. of So. Car.	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.
SNIDER, Howard Roy	U. of Oregon	MD 1950	Mpls. Gen. Hospital, Minneapolis, Minn.
SOSHEA, John William	Northwestern U.	MB 1949 MD 1950	102 110 2nd Ave. S.W., Rochester, Minn.
TEIPNER, William Albert	U. of Oregon	MD 1950	Mpls. Gen. Hospital, Minneapolis, Minn.
WHITMER, John Harrison	U. of Oregon	MD 1950	315 Wing Bldg., Grants Pass, Ore.

Reciprocity Candidates

Name	*School*		*Address*
BLOOD, Arthur McCrary	Emory U.	MD 1945	102 110 2nd Ave. S.W., Rochester, Minn.
BRIDGE, Ezra Volk	Cornell U.	MD 1940	Mineral Springs San., Cannon Falls, Minn.
BURLESON, Robert Joe	U. of Louisville	MD 1943	102 110 2nd Ave. S.W., Rochester, Minn.
COUGHLIN, Bertrand Daniel	St. Louis U.	MD 1931	1829 Med. Arts Bldg., Minneapolis, Minn.
CRAIG, Joseph Deafner	Ohio State U.	MD 1947	102 110 2nd Ave. S.W., Rochester, Minn.
CROMARTIE, William James	Emory U.	MD 1937	224 Millard Hall, University of Minn., Minneapolis, Minn.
DAVIS, Neal Balbach	U. of Nebraska	MD 1950	102 110 2nd Ave. S.W., Rochester, Minn.
ERICKSON, George Philip	Creighton U.	MD 1949	2229 3rd Ave. E., Hibbing, Minn.
FORNEY, William Dwight	U. of Chicago	MD 1948	102 110 2nd Ave. S.W., Rochester, Minn.
FRAZIER, Shervert Hughes	U. of Illinois	MD 1943	102 110 2nd Ave. S.W., Rochester, Minn.
GREEN, Paul Anderson	U. of Oklahoma	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.
HENDERSON, Charles Carey	Southwestern U.	MD 1945	102 110 2nd Ave. S.W., Rochester, Minn.
HILDEBRAND, John Edward	Wayne U.	MD 1944	502 Beltrami, Bemidji, Minn.
JACKSON, Robert Toombs	U. of Georgia	MD 1941	102 110 2nd Ave. S.W., Rochester, Minn.
JACOB, Jr., Norman Henry	U. of Texas	MD 1945	U. of M. Hospitals, Minneapolis, Minn.
KEMP, Jr., Robert Christie	Tulane U.	MD 1935	1829 Med. Arts Bldg., Mineapolis, Minn.
KLAKEG, Clayton Harold	Temple U.	MD 1945	102 110 2nd Ave. S.W., Rochester, Minn.
MENGES, Charles George Henry	Temple U.	MD 1943	102 110 2nd Ave. S.W., Rochester, Minn.
NORRIS, Lewis Duroc	U. of Iowa	MD 1943	1009 Nicollet Ave., Minneapolis, Minn.
ROBERTS, Stanley Eugene	Washington U.	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.
SULLIVAN, Charles Roger	U. of Rochester	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.
SWAIN, Francis M.	U. of Louisville	MD 1942	1210 Lowry Med. Arts Bldg., St. Paul, Minn.

PHYSICIANS LICENSED

National Board Candidates

			Address
FRY, Donald Lewis	Harvard U.	MD 1949	Vet. Adm. Hospital, Minneapolis, Minn.
HABEIN, Jr., Harold Clinton	U. of Minnesota	MB 1946 MD 1950	102 110 2nd Ave. S.W., Rochester, Minn.
HARDY, William McClure	George Washington U.	MD 1949	102 110 2nd Ave. S.W., Rochester, Minn.
JOHNSTON, Edward Vaughan	Northwestern U.	MB 1946 MD 1947	102 110 2nd Ave. S.W., Rochester, Minn.
SHAFER, Charles Leon	Harvard U.	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.
VIVIAN, Donald Norman	Boston U.	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.

PHYSICIANS LICENSED JULY 20, 1951

June 1951 Examination

Name	*School*		*Address*
AGER, Ernest Arthur	U. of Minnesota	MB 1951	St. Mary's Hospital, Duluth, Minn.
AHOLA, Kenneth Emil	U. of Minnesota	MB 1951	Mpls. Gen. Hospital, Minneapolis, Minn.
ALEXANDER, Carl Stuart	Columbia U. Col. of Phy. & Surg.	MD 1948	U. of M. Hospitals, Minneapolis, Minn.
ANDERSON, John William	U. of Minnesota	MB 1951	Ancker Hospital, St. Paul, Minn.
ANDERSON, Wallace Everett	U. of Minnesota	MB 1951	Ancker Hospital, St. Paul, Minn.
BAER, Walter	Albert Ludwigs U. Freiburg, Germany	MD 1921	814 Lowry Med. Arts Bldg., St. Paul Minn.
BERGE, Kenneth George	U. of Minnesota	MB 1951	Boston City Hospital, Boston, Mass.
BERKAS, Ernest Maurice	U. of Minnesota	MB 1951	St. Joseph's Hospital, St. Paul, Minn.
BLANCHARD, Russell Stanfield	U. of Minnesota	MB 1951	St. Barnabas Hosp., Minneapolis, Minn.
BOLSTAD, Owen C.	U. of Minnesota	MB 1951	St. Luke's Hospital, Duluth, Minn.
BOLTER, Arthur	U. of Minnesota	MB 1951	Sacramento Co. Hosp., Sacramento, Cal.
BROKKEN, Jack Ridley	U. of Minnesota	MB 1951	Mercy Hospital, San Diego, Calif.
CANNON, Thomas Patrick	U. of Minnesota	MB 1951	Letterman Army Hospital, San Francisco, Calif.
CAREY, Jr., James Bain	U. of Minnesota	MB 1951	U. of M. Hospitals, Minneapolis, Minn.
COBB, Jr., Leonard Arthur	U. of Minnesota	MB 1951	U. of Iowa Hospital, Iowa City, Iowa
CUMMING, Edward Dale	U. of Minnesota	MB 1951	U. of M. Hospitals, Minneapolis, Minn.
DÖSCHERHOLMEN, Alfred	U. of Oslo, Norway	Master Art's in Med. 1946	Asbury Hospital, Minneapolis, Minn.
DURYEA, Jr., Willis Mortimer	U. of Minnesota	MB 1951	Ancker Hospital, St. Paul, Minn.
EDWARDS, Donna Mae	U. of Minnesota	MB 1951	2700 Utica Ave., St. Louis Park, Minneapolis, Minn.
ELI, Earl Wilbur	U. of Minnesota	MB 1951	King's Co. Hospital, Seattle, Wash.
ELROD, Calvin Roger	U. of Minnesota	MB 1951	King's Co. Hospital, Seattle, Wash.
ERLANSON, Andrew Cornell	U. of Minnesota	MB 1951	699 S. Snelling, St. Paul, Minn.
FRYDENLUND, Conrad Bernard	U. of Minnesota	MB 1951	San Bernardino Co. Hosp., San Bernardino, Calif.
GILL, Theodore Marshall	U. of Minnesota	MB 1951	Mpls. Gen. Hospital, Minneapolis, Minn.
HAGEN, John Doran	U. of Minnesota	MB 1949 MD 1950	Vet. Adm. Hospital, Minneapolis, Minn.
HALD, Christian Peter	U. of Minnesota	MB 1951	Santa Clara Co. Hosp., San Jose, Calif.
HALFHIDE, Rudolph A.	McGill U.	MD 1944	102 110 2nd Ave. S.W., Rochester, Minn.
HANSEN, Milo Luverne	U. of Minnesota	MB 1951	Ancker Hospital, St. Paul, Minn.
HANSON, Martin Everett	U. of Chicago	MD 1950	368 Forest Drive S. E., Cedar Rapids, Ia.
HEIMARK, John Jacob	U. of Minnesota	MB 1951	San Francisco Hosp., San Francisco, Cal.
HEISEL, John George	Marquette U.	MD 1950	1302 Tower Ave., Superior, Wis.
HENDRICKS, Curtis John	U. of Minnesota	MB 1951	Ancker Hospital, St. Paul, Minn.
HEPPER, Norman Gordon	U. of Illinois	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.
HERMAN, Bernard Edward	U. of Minnesota	MB 1951	Michael Reese Hospital, Chicago, Ill.
HOLLIFIELD, Guy Foster	U. of Virginia	MD 1950	Mpls. Gen. Hospital, Minneapolis, Minn.
HOLTER, Wallace Russell	U. of Minnesota	MB 1951	2701 Monterrey Ave., Minneapolis, Minn.
HOOPER, Worth A.	U. of Minnesota	MB 1951	San Diego City & County Hospital, San Diego, Calif.
HOULTON, William Henry	U. of Minnesota	MB 1951	Mpls. Gen. Hospital, Minneapolis, Minn.
HUFFINGTON, Herb Leonard	U. of Minnesota	MB 1951	Santa Clara Co. Hosp., San Jose, Calif.
JACKSON, Richard Lewis	Marquette U.	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.
JAHNKE, William Robert	Temple U.	MD 1949	U. S. Navy Recruiting Station, Federal Office Bldg., Minneapolis, Minn.
JOHNSON, Thomas Stanley	U. of Minnesota	MB 1951	U. S. Naval Hosp., Bethesda, Md.
KELLEY, Roger Edwin	U. of Minnesota	MB 1951	Henry Ford Hospital, Detroit 2, Mich.
KELLY, Charles Francis	U. of Minnesota	MB 1951	4224 Cedar Ave., Minneapolis, Minn.
KNOEDLER, John Paul	Marquette U.	MD 1950	Vet. Adm. Hospital, Minneapolis, Minn.
KOSIAK, Jr., John	U. of Minnesota	MB 1950 MD 1951	c/o Postmaster, Interlaken, N. Y.
KRAFFT, Walter Edwin	U. of Minnesota	MB 1951	St. Luke's Hospital, Chicago, Ill.
KURTH, Milton Elmer	U. of Illinois	MD 1936	102 110 2nd Ave. S.W., Rochester, Minn.
LARSON, Lewis William	U. of Minnesota	MB 1951	Ancker Hospital, St. Paul, Minn.
LATTS, Elliot Mandel	U. of Minnesota	MB 1951	Phila. Gen. Hosp., Philadelphia, Pa.

LAWRENCE, Van Sheerin	U. of Minnesota	MB 1951	St. Joseph's Hospital, St. Paul, Minn.
LEE, Will Win	U. of Minnesota	MB 1951	Ancker Hospital, St. Paul, Minn.
LEVERSEE, John H.	U. of Minnesota	MB 1951	Harborview King Co. Hosp., Seattle, Wash.
LILLEHEI, Richard Carlton	U. of Minnesota	MB 1951	Mpls. Gen. Hospital, Minneapolis, Minn.
LOFGREN, Eric Paul	U. of Illinois	MD 1944	102 110 2nd Ave. S.W., Rochester, Minn.
LUND, Anthony James	U. of Illinois	MD 1950	Starekow Clinic, Thief River Falls, Minn.
LYONS, Henry Richard	Georgetown U.	MD 1945	102 110 2nd Ave. S.W., Rochester, Minn.
MacDONALD, William John	U. of Alberta	MD 1945	102 110 2nd Ave. S.W., Rochester, Minn.
McKELVIE, Allan McNeill	U. of Glasgow, Scotland	MB Ch. B. 1939	1801 K St. N.W., Washington, D. C.
McLINDEN, John Warren	U. of Minnesota	MB 1951	Santa Clara Co., Hosp., San Jose, Calif.
MESSENHEIMER, Myron G.	U. of Kansas	MD 1939	Student Health Service, University of Minn., Minneapolis, Minn.
MIDBOE, Gilbert Theodor	U. of Minnesota	MB 1951	Ancker Hospital, St. Paul, Minn.
MIDDLEBROOK, John Edward	U. of Minnesota	MB 1951	San Francisco City Hosp., San Francisco, Calif.
MILLER, George Emerson	U. of Alberta, Can.	MD 1945	102 110 2nd Ave. S.W., Rochester, Minn.
MILLER, Herman	U. of Minnesota	MB 1951	Ancker Hospital, St. Paul, Minn.
MITCHELL, Russell Harry	U. of Minnesota	MB 1951	Gorgas Hospital, Ancon, Panama Canal
MORTENSON, Howard Odell	U. of Minnesota	MB 1951	Tripler Army Hospital, Moanalua, Oahu, T. H.
NEIBERGS, Pauls	U. of Latvia Riga, Latvia	"Physician" 11-5-38	St. Joseph's Hospital, St. Paul, Minn.
NORQUIST, Stanley Roy	U. of Minnesota	MB 1951	814 4th St. S.E., Minneapolis, Minn.
OGDEN, Harry Sweet	Northwestern U.	MB 1948 MD 1949	University of Minn. Hospitals, Dept. Rad., Minneapolis, Minn.
O'NEILL, Harold Patrick	U. of Minnesota	MB 1951	Rochester Gen. Hosp., Rochester, N. Y.
OWEN, William Vaughan	U. of Minnesota	MB 1951	Rochester Gen. Hosp., Rochester, N. Y.
PETTERSEN, George Richard	U. of Minnesota	MB 1951	2123-A Folwell St., St. Paul, Minn.
PEW, Wilmer Laurence	U. of Minnesota	MB 1951	U. of M. Hospitals, Minneapolis, Minn.
POLLAK, Kurt	U. of Minnesota	MB 1951	Mpls. Gen. Hospital, Minneapolis, Minn.
PRENTICE, Bruce Culver	U. of Minnesota	MB 1951	2146 Hartford, St. Paul, Minn.
RACER, Harley Joseph	U. of Minnesota	MB 1951	2135-A Folwell St., University Grove E., St. Paul, Minn.
RAJALA, Arnold Ivar	U. of Minnesota	MB 1951	Mpls. Gen. Hospital, Minneapolis, Minn.
RATELLE, Alexander Eugene	U. of Minnesota	MB 1951	St. Joseph's Hospital, St. Paul, Minn.
RAYNER, Ralph Reuben	U. of Minnesota	MB 1951	Mpls. Gen. Hospital, Minneapolis, Minn.
ROBERTS, Byron Harvey	U. of Minnesota	MB 1951	Mpls. Gen. Hospital, Minneapolis, Minn.
ROREM, Joseph Andrew	U. of Minnesota	MB 1951	2081-E Univ. Grove E., St. Paul, Minn.
ROSENBERG, Robert Harris	U. of Minnesota	MB 1951	605 Minor Ave., Seattle, Wash.
SADOFF, Charles Nathan	U. of Minnesota	MB 1951	St. Joseph's Hospital, St. Paul, Minn.
SCHISSEL, Gregory Anthony	Creighton U.	MD 1951	Ancker Hospital, St. Paul, Minn.
SCHNEIDER, Constantine Otto	U. of Oregon	MD 1950	2515 N. Killingsworth, Portland, Ore.
SCHNELL, Frederick Sherman	Marquette U.	MD 1950	East Range Clinic, Virginia, Minn.
SISSON, Norvel Dale	U. of Minnesota	MB 1951	St. Joseph's Hospital, St. Paul, Minn.
SMILEY, Donald Pearce	U. of Chicago	MD 1950	182 Donny De, Huntlee Village, Algiers, Louisiana
SPENCER, Lorin Clair	U. of Minnesota	MB 1951	2701 14th Ave., Oakland, Calif.
SQUIRE, George Varnum	U. of Minnesota	MB 1951	Emanuel Hospital, Portland, Ore.
STANCHFIELD, Robert Franklin	U. of Minnesota	MB 1951	624 8th Ave. S.E., Minneapolis, Minn.
SWENSON, Donald Bjork	U. of Minnesota	MB 1951	Ancker Hospital, St. Paul, Minn.
TEBERG, Annabel Jean	U. of Minnesota	MB 1951	Los Angeles City & County Hospital, Los Angeles, Calif.
VISSCHER, Barbara R.	U. of Minnesota	MB 1951	San Francisco Hosp., San Francisco, Cal.
WEXLER, Harold M.	U. of Minnesota	MB 1951	Michael Reese Hospital, Chicago, Ill.
WEYHRAUCH, William Roger	U. of Minnesota	MB 1951	St. Luke's Hospital, Chicago, Ill.
WHITE, Eugene Leslie	U. of Minnesota	MB 1951	Tripler Army Hospital, APO 438, San Francisco, Calif.
WILD, John Julian	U. Col. Hospital, U. of London, Eng.	MB and Ch. B. 1942	University of Minn., Dept. Elec. Engr., Minneapolis, Minn.
WILSON, Louis James	U. of Minnesota	MB 1951	1292 Maynard Dr., Apt. 333, St. Paul, Minn.
WOHLRABE, Donald Edwin	U. of Minnesota	MB 1951	Phila. Gen. Hospital, Philadelphia, Pa.
WRIGHT, Kermit Joseph	U. of Minnesota	MB 1951	Mpls. Gen. Hospital, Minneapolis, Minn.
YEOMANS, Merrill Brooks	U. of Minnesota	MB 1951	U. Texas Hospital, Galveston, Texas

Reciprocity Candidates

Name	*School*		*Address*
CHAVEZ, Demetrio Armijo	U. of Colorado	MD 1949	2726 E. 50th St., Minneapolis, Minn.
CHILES, Noah Hampton	U. of Louisville	MD 1944	102 110 2nd Ave. S.W., Rochester, Minn.
FEINBERG, Philip	U. of Minnesota	MB 1938 MD 1939	1127 Med. Arts Bldg., Minneapolis, Minn.
HOEFLICH, Bert Joseph	Baylor U.	MD 1946	953 Med. Arts Bldg., Minneapolis, Minn.
KEENAN, Hugh Carter	Marquette U.	MD 1944	102 110 2nd Ave. S.W., Rochester, Minn.
LAW, Harrison Edwin	Col. Phy. & Surg., Columbia U.	MD 1938	Lenont-Peterson Clinic, Virginia, Minn.

MacKINNON, Hector Neil	U. of Alberta	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.
MINNO, Alexander Mackey	U. of Pittsburgh	MD 1947	102 110 2nd Ave. S.W., Rochester, Minn.
NYWALL, Dean David	U. of Nebraska	MD 1950	Slayton Clinic, Slayton, Minn.
SCHNECK, Jack Irving	Baylor U.	MD 1937	2215 Plymouth Ave. N., Minneapolis, Minn.
SMITH, Roger Carlton	U. of Indiana	MD 1944	102 110 2nd Ave. S.W., Rochester, Minn.
SPRAFKA, Gregory Anthony	St. Louis U.	MD 1950	526 Hamm Bldg., St. Paul, Minn.
TAYLOR, Edwin Addison	Col. of Med. Evang.	MD 1944	102 110 2nd Ave. S.W., Rochester, Minn.
WENTZLER, James Donald	Jefferson U.	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.
WESTBY, Norval Martin	U. of Nebraska	MD 1950	Madison, Minn.

National Board Candidates

Name	*School*		*Address*
CHRISTENSEN, John Raymond	U. of Iowa	MD 1947	205 W. Second St., Duluth, Minn.
ERICKSON, Robert Lyle	Geo. Washington U.	MD 1950	190 Main St., Caledonia, Minn.
LOVELACE, Jr., Daniel Dudley	Tufts Col.	MD 1934	401-5 First St., Virginia, Minn.
WESTBY, Norval Martin	McGill U.	MD 1949	4107 Woodland Ave., Duluth, Minn.

PHYSICIANS LICENSED NOVEMBER 9, 1951

October 1951 Examination

Name	*School*		*Address*
BARR, James S.	U. of Minnesota	MB 1951	Ancker Hospital, St. Paul, Minn.
BENOIT, Clair Francis	U. of Manitoba, Can.	MD 1943	102 110 2nd Ave. S.W., Rochester, Minn.
GRANT, Suzanne	U. of Minnesota	MB 1949 MD 1950	4715 First Ave. So., Minneapolis, Minn.
HARE, Robert Lewis	U. of Illinois	MD 1949	102 110 2nd Ave. S.W., Rochester, Minn.
HOLT, Prescott Brooks	Columbia U.	MD 1948	102 110 2nd Ave. S.W., Rochester, Minn.
KELLEY, Walter Maynard	U. of Wisconsin	MD 1950	722 E. Second St., Duluth, Minn.
KELLY, Robert Thomas	U. of Minnesota	MB 1951	Mpls. Gen. Hospital, Minneapolis, Minn.
KOHLHASE, Robert Edward	U. of Minnesota	MB 1951	Indianapolis Gen. Hosp., Indianapolis, Ind.
LYONS, James Henry	U. of Chicago (Rush)	MD 1942	Wayne Co. Gen. Hosp., Eloise, Mich.
McQUAY, Norman William	Queen's U., Can.	MD 1944	102 110 2nd Ave. S.W., Rochester, Minn.
MOLENAAR, Robert Eugene	U. of Minnesota	MB 1951	Ancker Hospital, St. Paul, Minn.
MOORES, Kenneth Dabney	U. of Oregon	MD 1950	102 110 2nd Ave. S.W., Rochester, Minn.
NEIBERGS, Lidija Irma	U. of Latvia Riga, Latvia	"Physician" 10-25-39	539 Capitol Blvd., St. Paul, Minn.
RABENS, Robert Allen	U. of Illinois	MD 1950	102 110 2nd Ave. S.W., Rochester, Minn.
ROBINSON, John Gates	U. of Toronto, Can.	MD 1949	102 110 2nd Ave. S.W., Rochester, Minn.
SHEA, Samuel Edward	Georgetown U.	MD 1946	Vet. Adm. Hospital, Minneapolis, Minn.
SIDELL, Franklin DuBois	U. of Chicago	MD 1950	102 110 2nd Ave. S.W., Rochester, Minn.
SMITH, William George	U. of Chicago	MD 1947	102 110 2nd Ave. S.W., Rochester, Minn.
SPANG, William Matthew	Marquette U.	MD 1950	1930 Superior St. W., Duluth, Minn.
STEVENS, Grant Melvin	U. of Kansas	MD 1950	102 110 2nd Ave. S.W., Rochester, Minn.
ULERY, Richard Myron	Johns Hopkins U.	MD 1950	102 110 2nd Ave. S.W., Rochester, Minn.
WHITE, Roy Alan	U. of Washington	MD 1950	102 110 2nd Ave. S.W., Rochester, Minn.
WITTCHOW, Allen William	U. of Wisconsin	MD 1950	102 110 2nd Ave. S.W., Rochester, Minn.

Reciprocity Candidates

Name	*School*		*Address*
ARDAN, Jr., Nicholas Ivan	Med. Col. of Va.	MD 1950	102 110 2nd Ave. S.W., Rochester, Minn.
BABCOCK, Oliver Lemont	Temple U.	MD 1946	Delano, Minn.
BAIRD, Raymond Lynn	U. of Colorado	MD 1950	287 N. Smith Ave., St. Paul, Minn.
BELL, Jr., Harry Manning	Syracuse U.	MD 1944	1201 E. Third St., Duluth, Minn.
BRZICA, Stephen Marion	U. of Nebraska	MD 1950	Worthington, Minn.
BURT, Robert Raymond	U. of Oregon	MD 1944	102 110 2nd Ave. S.W., Rochester, Minn.
CLARK, Doyce McIntire	Baylor U.	MD 1938	102 110 2nd Ave. S.W., Rochester, Minn.
COFER, Jr., Vernon Lonsdale	Med. Col. of Va.	MD 1948	102 110 2nd Ave. S.W., Rochester, Minn.
DOMZALSKI, Casimir Anthony	U. of Michigan	MD 1945	102 110 2nd Ave. S.W., Rochester, Minn.
DUNN, Jr., Jack	U. of Texas	MD 1948	102 110 2nd Ave. S.W., Rochester, Minn.
ERWIN, Paul Dair	U. of Oklahoma	MD 1946	U. of M. Hospitals, Minneapolis, Minn.
GATCHELL, Frank Gaffney	U. of Oklahoma	MD 1948	102 110 2nd Ave. S.W., Rochester, Minn.
GILLEY, Edwin Wayne	U. of Virginia	MD 1948	102 110 2nd Ave. S.W., Rochester, Minn.
GREEN, Jr., Robert Castleman	U. of Virginia	MD 1947	102 110 2nd Ave. S.W., Rochester, Minn.
KOPECKY, Frank Anthony	Creighton U.	MD 1948	102 110 2nd Ave. S.W., Rochester, Minn.
KROLL, Harry Gardner	U. of Chicago	MD 1950	102 110 2nd Ave. S.W., Rochester, Minn.
LESCOE, Richard John	U. of Pittsburgh	MD 1949	102 110 2nd Ave. S.W., Rochester, Minn.
LITZOW, Thaddeus Joseph	Marquette U.	MD 1949	102 110 2nd Ave. S.W., Rochester, Minn.
MEARS, Thomas Wakefield	Geo. Washington U.	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.

PHYSICIANS LICENSED

O'KEEFE, Jr., Mathew E.	Med. Col. of Va.	MD 1950	102 110 2nd Ave. S.W., Rochester, Minn.
OXFORD, Marvin Bradfield	U. of Texas	MD 1942	102 110 2nd Ave. S.W., Rochester, Minn.
PETTET, John Richard	U. of So. Calif.	MD 1949	102 110 2nd Ave. S.W., Rochester, Minn.
POWELSON, Myron Hopkins	U. of Louisville	MD 1949	102 110 2nd Ave. S.W., Rochester, Minn.
SLOSS, Pierce Thomas	U. of Iowa	MD 1947	102 110 2nd Ave. S.W., Rochester, Minn.
WEAVER, Rex Winfield	U. of Nebraska	MD 1950	102 110 2nd Ave. S.W., Rochester, Minn.
WISE, Jr., Fred Eugene	Med. Col. of Va.	MD 1945	102 110 2nd Ave. S.W., Rochester, Minn.

National Board Candidates

Name	*School*		*Address*
BLANK, Leo	Med. Col. of Va.	MD 1949	University of Minn., Dept. Rad., Minneapolis Minn.
CARUOLO, Joseph Emilio	Tufts Med. Col.	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.
CONWAY, Robert F.	U. of Minnesota	MB 1948 MD 1949	628 Elm St., St. Peter, Minn.
GOSSEL, John Dietrich	New York Med. Col.	MD 1950	102 110 2nd Ave. S.W., Rochester, Minn.
HALPIN, Mary Aiden	Tufts Med. Col.	MD 1950	102 110 2nd Ave. S.W., Rochester, Minn.
KILBY, Ralph Allen	U. of Buffalo	MD 1948	102 110 2nd Ave. S.W., Rochester, Minn.
KUHN, Arthur J.	U. of Cincinnati	MD 1949	102 110 2nd Ave. S.W., Rochester, Minn.
LASSETTER, Edgar Lovelace	McGill U., Can.	MD 1948	102 110 2nd Ave. S.W., Rochester, Minn.
LEV, Ralph	Jefferson Med. Col.	MD 1948	102 110 2nd Ave. S.W., Rochester, Minn.
MEDOFF, Alvin Sherwood	N. Y. Med. Col.	MD 1948	102 110 2nd Ave. S.W., Rochester, Minn.
PARK, Wilford Edison	U. of Toronto, Can.	MB 1927 MD 1940	1804 Humboldt Ave. So., Minneapolis,
PETERS, Jr., William Francis	Temple U.	MD 1949	102 110 2nd Ave. S.W., Rochester, Minn.
RAWLS, Thompson Townsend	Harvard U.	MD 1949	102 110 2nd Ave. S.W., Rochester, Minn.
SMITH, Ballard Franklin	U. of Buffalo	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.
SPELLMAN, Mitchell Wright	Howard U.	MD 1944	University of Minn., Dept. Surgery, Minneapolis, Minn.
STRUTHERS, Alfred Morgan	Syracuse U.	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.
TUERK, Milton	New York U.	MD 1950	102 110 2nd Ave. S.W., Rochester, Minn.
WEISMAN, Philip Albert	U. of Cincinnati	MD 1945	102 110 2nd Ave. S.W., Rochester, Minn.
WHITE, Philip Taylor	Geo. Washington U.	MD 1946	102 110 2nd Ave. S.W., Rochester, Minn.
WILSON, Jr., Leo Hughes	Duke U.	MD 1947	102 110 2nd Ave. S.W., Rochester, Minn.
WINKELMANN, Richard Knisely	Marquette U.	MD 1948	102 110 2nd Ave. S.W., Rochester, Minn.

SUMMARY OF A MESSAGE FROM THE BOARD OF TRUSTEES OF THE AMERICAN MEDICAL ASSOCIATION

D. W. LARSON, M.D.

Bismarck, North Dakota

Dr. Larson brought greetings from the Board of Trustees of the American Medical Association. He reported that the National Education Campaign accomplished much, concluding that this is the first time in the public relations advertising history, that a profession, business or industry has been successful in rallying nationwide advertising support; that this support endorsing medicine's fight against socialized medicine did not come from big business and reactionary forces, but from small advertisers on main street; that there were a large number of tie-in ads sponsored by state and county societies whose members had already contributed to the campaign; and that the results of the election show the impact of the doctors' influence on the local level.

Dr. Larson felt that the problems of immediate concern to the Board of Trustees are federal aid to medical education and clarification of the policy for public health units.

He said that the American Medical Association is opposed to federal aid to medical education because there is a lack of evidence that federal aid is the only solution, because provision of federal aid would very likely lead to federal controls, because it is believed that it would reduce the incentive to support schools on local levels, and because increased federal expenditures for federal aid to medical education would perpetuate higher taxes and the cycle would perpetuate itself. He asked, "If medical schools are subsidized, would they be dropped when peace comes? How will controls be avoided?"

Dr. Larson urged clarification of the AMA's stand on public health units. He advocated a clearer definition of public health and the practice of medicine. He said that not enough doctors belong to and are active in the Public Health organizations.

Dr. Larson declared that the American Medical Association belongs to you and to me—it is not only at 535 North Dearborn in Chicago. Citing it as a federacy of constituent associations, Dr. Larson explained that the House of Delegates is the legislative body, with the Board of Trustees handling the business side. He told doctors that it is very important to the House of Delegates that you send good men who will deliberate well on these things.

He urged frequent re-examination of the 12-point program of the American Medical Association. We, as doctors in America, he stated, will legislate and operate AMA as we want it done.

Dr. Larson asked, "Are we in tune with the social revolution that is going on in this country?" Any changes that we make in the future must be made with this policy in mind—the continuance of the free practice of medicine, he concluded.

Dr. Larson is a member of the Board of Trustees of the American Medical Association.

Minneapolis Surgical Society

Meeting of March 8, 1951

The President, HARVEY NELSON, M.D., in the Chair

A MODIFIED AMPUTATION OF THE THIGH—LATERO-MEDIAL FLAP

VIRGIL J. P. LUNDQUIST, M.D.

Minneapolis, Minnesota

Since amputation is known to be one of the oldest of surgical procedures, it is fitting that it be reconsidered by surgeons at times. Even though amputation may be overshadowed by some of the more complicated of modern surgical procedures, any new concept in its performance is worthy of consideration.

It has been demonstrated by archeological evidence that amputation was most likely known and practiced in the Neolithic Period of man's development. Stone and bone knives and saws of this period have been recovered which could well have been used for amputating extremities. Hippocrates, in the latter part of the 5th Cenutry B.C. recommended that gangrenous limbs be amputated through the upper area of gangrene. Celsus, at the beginning of the Christian Era, during the reign of Augustus Caesar, was one of the first to practice amputation through sound tissue above the area of gangrene.

During these early amputations, hemorrhage was of necessity one of the most deterring factors in carrying out the procedure. Celsus did advocate the ligation of vessels but the technique of ligature was not universally accepted until it was again reintroduced by the French Army Surgeon, Paré, in the 16th century. Prior to this time the management of hemorrhage was by cautery, burning oil, and pressure dressings.

This remained the point of progress in the field of amputation until the introduction of methods to obtain better amputation stumps. Lowdham was one of the first on record to advocate the use of skin flaps which were fashioned at the time of operation to serve as protection for the amputation stump. With the introduction of skin flaps, several names appear on the scene of amputation development among which may be mentioned those of Hey, Langenbeck, Liston, Teale, and Syme. A discussion of amputation development would not be complete without at least mention of the names of Baron Larrey, Napoleon's great Army Surgeon, and G. J. Guthrie of the British Medical Corps. Larrey made a tremendous contribution to surgery when he introduced his technique of debridement not unlike that practiced today. With the development of chemotherapy and antibiotics, amputation took on a new role. There has been a commendable tendency to conservatism. The guillotine method of amputation, so often used in the past, has been relegated to a position of lesser importance—and rightly so. Nevertheless, there are still real indications for its use.

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Guillotine Amputation

It is to a modified method of guillotine amputation that this presentation is directed. This modification is one which has been employed at the U. S. Veterans Administration Hospital in Minneapolis, Minnesota. In brief, it is a modification of the method wherein lateral and medial flaps of skin, fascia and muscle are retained to enable easier and more rapid healing of the stump and to eliminate to a large extent the secondary operative procedures so often necessary in the revision of the classical guillotine stump. With permission we can give the concept a name—The Latero-Medial Flap Amputation. Four aspects of the problem have been chosen to facilitate the presentation of this material. These will be discussed under the following: (1) indications; (2) technique employed; (3) advantages of the method; and (4) a statistical analysis.

1. *Indications.*—It may be mentioned that amputation is indicated to save life, to control the spread of infection, to combat the spread of disease, and to remove useless extremities.

It is extremely difficult to advance general rules for the employment of any surgical procedure. Without mention, it is understood that individual cases require individual considerations. Gangrene, however, is accepted as a major indication for amputation. It is perhaps the most frequent indication for amputation in the civilian practice of surgery. Grangrene has been the outstanding indication for the procedure herein described. To facilitate its consideration there has been a subdivision to include the peripheral vascular disorders predisposing to the gangrene, namely—arteriosclerosis obliterans—without diabetes and with diabetes; thromboangiitis obliterans (Buerger's disease); acute thrombosis; and acute embolism.

If we consider for a moment the pathogenesis of gangrene, the indication for amputation is self-evident in an arteriosclerotic obliterating process. Since the

blood supply is in the form of end arteries to the terminal parts and, in gangrene, thrombosis of these vessels occurs, there is little likelihood of there being adequate collateral supply as the collateral vessels are similarly involved in the generalized sclerotic process. The gangrene which follows is an attempt by "Nature" to slough the tissue which has been deprived of its blood supply. Sloughing is accomplished by an inflammatory process of destruction through the muscle, tendon, bone, subcutaneous tissue, and skin. There must be a good blood supply to the proximal tissues to effect this separation. In the presence of diseased vessels in the adjacent tissues, this good blood supply is lacking, the inflammatory reaction extends further proximalward, further thrombosis occurs, and the gangrene spreads. The products of dead and dying tissue resulting from this anemic aseptic necrosis are absorbed into the circulation and may lead to irreversible changes in an already aged and damaged myocardium or renal parenchyma. This progression of the process of the gangrene plus the added ever-present specter of infection with gas bacillus, tetanus, or other septic organism often casts amputation in a life-saving role.

This leads us to the second point under indication for the latero-medial flap procedure—when a guillotine type of amputation is desired. This implies the condition encountered in the patient debilitated by chronic disease, a patient in a precarious state due to an underlying cardiovascular or renal disease, often under intensive medical management for this underlying disease when the acute episode of thrombosis or embolism strikes, and as a result a patient who is an extremely poor surgical risk. For these reasons a procedure which can be done rapidly, which does not consume time in creating flaps and thereby does not expose the patient to the added risks of a prolonged operation and anesthesia, is definitely indicated. Then, further, the guillotine type of open or semi-open amputation may be indicated in the case of an infected extremity. When there is a question as to whether the infection is under control or not and when the amputation is done as an adjunct to the control of the infection or the underlying disease as in the case of diabetic gangrene, open amputation is of value. There is also the question which may arise as to whether the chosen level is one of viability and whether a definitive procedure at this level will be successful. Often with these presenting circumstances a guillotine open type of amputation is to be commended.

For our last consideration under indications, it has been chosen to restrict this procedure to the amputations at low thigh level. The type of scar which results as it does running anteroposteriorly around the stump does not provide for a good stump in the lower leg. The retraction of the scar may prove troublesome between the two bones of the lower leg, and this may lead to a very unsatisfactory stump. The maximum length of the stump in the thigh amputation is to be recommended for a better functioning stump and better wearing of a prosthesis. Since the real indication for this procedure is in peripheral vascular disorders and since these most often lead to a lower extremity amputation, it is thought that the low thigh level should be indicated as the elected site of amputation when this procedure is employed. It is further known that when the low thigh level is elected for this procedure, a satisfactory amputation stump does result.

2. *Technique Employed.*—As a preliminary to presenting points in the actual technique peculiar to this procedure, it may be stated that the patient is prepared, and draped in the usual manner. The extremity is sterilly wrapped so that it can be manipulated during the operation by the surgeon in the operative field. As to the choice of anesthetic agent, Baird's solution (sodium pentothal and tubocurare) has been employed. There is nothing in the method itself to preclude the use of any one anesthetic agent. A tourniquet has not been used since this amputation has been done in patients with an already compromised peripheral circulation.

This has been done as a guillotine type of procedure. The long amputation knife is the instrument used. The flaps are cut from the inside outwardly in two bold strokes. The amputation knife is thrust into the thigh at a selected point on the anterior surface of the thigh in the midline. This point is about 3 to 4 inches above the knee joint. The knife is advanced to the femur and it is then forced laterally hugging the lateral surface of the femur until it passes the extent of the bone. An attempt is then made to have the tip of the knife emerge through the skin of the posterior thigh in the midline. It is thrust completely through the thigh. The lateral flap of muscle, fascia, subcutaneous tissue and skin is then fashioned by drawing the knife outwardly and downward toward the knee joint. This skirts the flare of the femur and is made by passing the knife outwardly at approximately a 45 degree angle. A similar type of cut is then made on the medial side of the thigh attempting to have the two flaps join at the apex on both the anterior and the posterior surface of the thigh. The hemorrhage is of course controlled following the two strokes. The larger vessels are usually transected with the medial cut. The hemorrhage is usually not profuse since these patients have a deficient peripheral circulation and it is not uncommon to find the main arterial channels completely obliterated.

The bone is divided at the apex of the incision. This is done by incising the periosteum circularly around the shaft of the femur and making the saw cut in the bone in the same line as the periosteum. By not stripping the periosteum proximalward it is thought that the formation of ring sequestra is avoided.

In the control of the hemorrhage and the achievement of hemostasis, chromic catgut is used as ligature material. The nerve is transected cleanly, using a razor blade or sharp scalpel blade and if necessary is ligated with a non-absorbable ligature. This is applied in an attempt to avoid cutting through the perineurium. This may help in the prevention of the development of painful neuromata.

The point of modification of the guillotine principle then arises. After the tissues have been divided and their appearance noted, one is in a better position to

evaluate their viability. If it appears that the blood supply is fully adequate and there is no question as to the viability, one may elect to close the flaps at least partially. This is one of the real advantages of the procedure. The flaps may be closed beginning at the anterior aspect without compromising the drainage. The cleft created by the lateral and medial flaps leaves a very adequate drainage site posteriorly. This is in the ideal position for maximum dependent drainage with the patient lying in bed on his back. In the suture of the flaps it is only necessary to approximate the skin with a few silk sutures placed interruptedly carrying the closure as far posteriorly as is elected. If the closure is quite complete a drain is brought out at the posterior angle. Occasionally it is necessary to trim the skin edge posteriorly at the apex if the two cuts found the knife tip leaving a bridge of skin between them.

The dressing which has been used has been one fashioned from stockinette. A voluminous gauze dressing is applied to the stump. If it has been elected to leave the flaps open, vaseline gauze strips are placed next to the raw surface. Following the gauze dressing, the stockinette of the proper size is rolled down over the dressing on the end of the stump. ACE adherent is then painted on over the stockinette to cause it to stick to the skin of the thigh. The stockinette is then secured by wrapping the thigh with an elastic bandage. This is wrapped down over the end of the stump and carried out for a short distance on the stockinette to cause the entire stump to assume a cone shape. A piece of rope is tied to the redundant portion of the stockinette and is attached to a 1 pound weight and brought over the end of the patient's bed on a pulley. This traction is maintained for a few days to maintain the stump in good position preventing contracture, to prevent the stump from becoming edematous and painful, and to prevent too marked retraction of the flaps from occurring.

3. *Advantages of the Method.*—To re-emphasize what are thought to be some of the advantages of this amputation modification, it should be mentioned that this is an amputation of the guillotine type and thus has the advantages incident to such a method. It can be performed very quickly thus eliminating the need for a prolonged procedure in an already debilitated patient. It can be modified to take advantage of the conditions encountered. If there is adequate viability and no contraindication, closure of the flaps may be elected even to primary closure with drainage at the posterior angle. This is probably one of the greatest advantages —the creation of a very adequate drainage channel at the most dependent posterior angle.

A further real advantage is attained in that it avoids a secondary operation to obtain a satisfactory stump in a high percentage of cases. The plastic closure of the classical guillotine stump is well known. The stump obtained with this procedure is very satisfactory even when there is no attempt made to close it at the initial operation. In the majority of cases there is no need for revision of the stump.

With this method, a very adequate stump is obtained for the wearing of the proper prosthesis. Since these patients have inadequate peripheral circulation, the indicated prosthesis for them is the ischial weight-bearing type. It is not recommended that these patients bear weight on the end of their stumps with the poor peripheral circulation they possess. The presence of the scar running anteroposteriorly around the end of the stump is therefore not a disadvantage. Several patients having had this type of amputation have at the present time satisfactorily worn their ischial weight-bearing prosthesis with no stump difficulties for periods up to three years.

4. *Statistical Analysis.*—During the period 1947 to 1950 this method of amputation was employed for twenty-three patients at the USVA Hospital at Minneapolis, Minnesota. Twenty-four amputations were done, in one instance a bilateral amputation was performed. In Table I, the conditions for which the amputations were done are listed.

TABLE I.

Diagnosis	Number of Cases
Arteriosclerosis obliterans	12
without Diabetes	6
with Diabetes	2
Thromboangiitis obliterans	7
Acute Thrombosis	4
Acute Embolism	—
Total	31

To clarify the total figure of thirty-one, it must be mentioned that six cases included in the arteriosclerosis obliterans group and 1 case in the thromboangiitis obliterans group are also classified with the acute thromboses. These were cases in which an intercurrent acute thrombosis occurred in the course of their underlying condition. Thus the original total of twenty-four cases is verified.

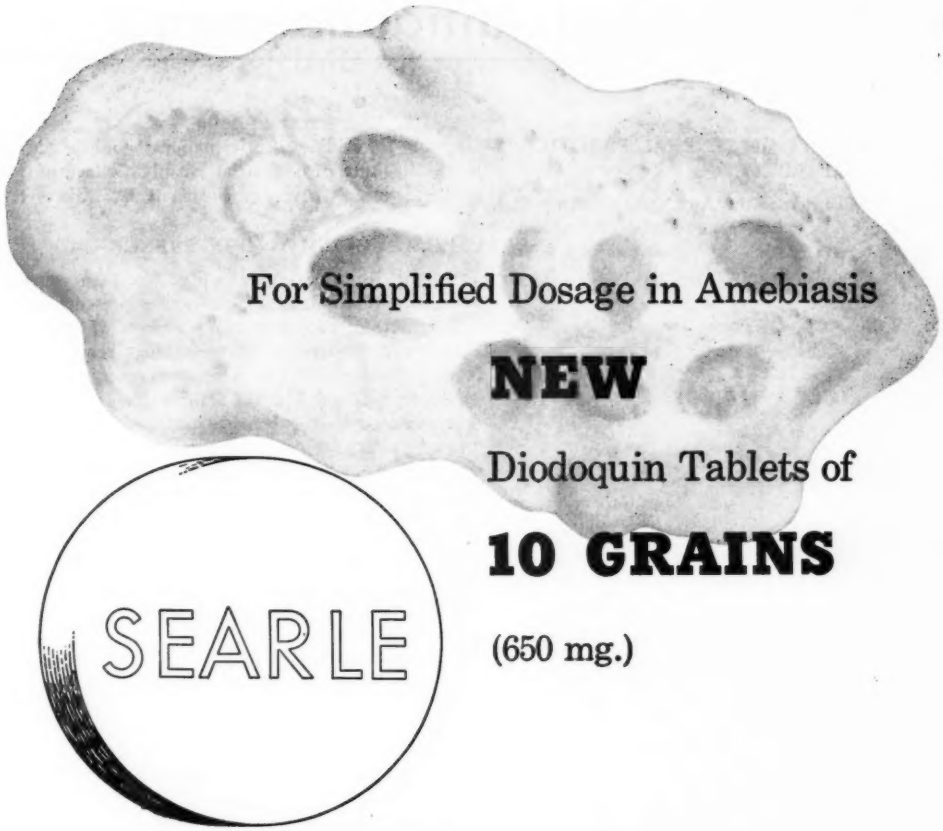
In Table II will be found an analysis of the results of healing in the cases under consideration.

TABLE II.

	Primary Healing	Prompt Healing	Delayed Healing	Requiring Revision
Arteriosclerosis obliterans:				
without Diabetes	5	5	1	1
with Diabetes	4		1	1
Thromboangiitis obliterans			1	1
Acute Thrombosis	3	3	1	
Acute Embolism (1 patient expired within 24 hours)	1		1	1
Total	13	8	5	4
Percentage	43% (10)	22% (5)	17% (4)	17%

In Table II, in arriving at an expression of the totals on a percentage basis, it was necessary to correct for the duplicated cases in the acute thrombosis group. The figures in the parentheses are the corrected totals and are used to arrive at the percentage figure. The patient who expired within 24 hours of operation is not in-

(Continued on Page 1224)



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Thus the twenty-day recommended dosage schedule is accomplished with a total of 60 instead of 200 tablets. The cost to the patient is reduced accordingly.

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(*diiodohydroxyquinoline*)

—is a well-tolerated, relatively nontoxic compound containing 63.9 per cent of iodine.

Now available in tablets of:

3.2 grains (210 mg.), bottles of 100 and 1,000
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Be sure to prescribe the 10 gr. (650 mg.) size for full adult dosage.

SEARLE RESEARCH IN THE SERVICE OF MEDICINE

✦ Reports and Announcements ✦

AMERICAN ACADEMY OF GENERAL PRACTICE MINNESOTA CHAPTER

The Minnesota Chapter of the American Academy of General Practice held its first postgraduate clinical session in Saint Paul on November 14. Physicians from throughout the nation were guest lecturers at the one-day meeting.

President of the state chapter is Dr. Albert E. Ritt, Saint Paul.

AMERICAN COLLEGE OF SURGEONS

At a meeting of the board of regents of the American College of Surgeons held at the Fairmont Hotel in San Francisco on November 9, Dr. Evarts A. Graham of St. Louis was appointed to succeed Dr. Arthur W. Allen of Boston as chairman. Dr. Graham is emeritus professor of surgery, Washington University School of Medicine and emeritus surgeon-in-chief, Barnes and St. Louis Children's Hospital. Dr. Graham has been a member of the board of regents since 1940.

Among the new fellows initiated at the San Francisco meeting were the following: Dr. Frank J. Ankner, Dr. Milton E. Baker, Dr. Norman B. Bloom, Dr. David Gaviser, Dr. Walter Lees Hoffman, Dr. Vernon L. Lindberg, Dr. Mancel T. Mitchell, Dr. Jacob H. Strickler, all of Minneapolis; Dr. Edward W. Sickels, Saint Paul; Dr. Oliver Howard Beahrs, Dr. Allan L. Haynes, Dr. John C. Ivins, Dr. Joseph M. Janes, all of Rochester; Dr. Wilford J. Dewesse, Bemidji; and Dr. William O. Finkelnburg, Winona.

FELLOWSHIPS IN INDUSTRIAL MEDICINE

The Institute of Industrial Health of the University of Cincinnati will accept applications for a limited number of fellowships offered to qualified candidates who wish to pursue a graduate course of instruction in preparation for the practice of industrial medicine. Any registered physician who is a graduate of a Class A medical school and who has completed satisfactorily at least two years of training in a hospital accredited by the American Medical Association may apply for a fellowship in the Institute of Industrial Health. (Service in the Armed Forces or private practice may be substituted for one year of training.)

The course of instruction consists of a two-year period of intense training in industrial medicine, followed by one year of practical experience under adequate supervision in industry. Candidates who complete satisfactorily the course of study will be awarded the degree of Doctor of Industrial Medicine.

During the first two years, the stipends for the Fellowship vary, in accordance with the marital status of the individual, from $2,100 to $3,000. In the third year the candidate will be compensated for his service by the industry in which he is completing his training. Requests for additional information should be addressed to the Institute of Industrial Health, College of Medicine, Eden and Bethesda, Cincinnati 19, Ohio.

CHICAGO CLINICAL CONFERENCE

The Clinical Conference, which has been established by the Chicago Medical Society for presentation each spring, offers lectures on many aspects of medicine to keep doctors abreast of the new things being developed from year to year. The next conference will be held March 4, 5, 6 and 7 in the Palmer House, Chicago.

In response to popular demand, there will be an increased number of demonstrations or workshop periods in addition to the regular series of lectures. These demonstrations include presentation of patients, carefully selected scientific movies, and other features interesting from an educational standpoint. The lectures are on subjects of interest to both the general practitioner and the specialist and will be one-half hour in duration. Scientific exhibits will present visually some of the most recent advances in medicine.

The completed program will be available shortly and will be mailed upon request.

SCHOOL OF CYTOLOGY

The Cancer Cytology Center of the Dade County Cancer Institute, an affiliate of the Medical Research Foundation of Dade County in Miami, Florida, has announced its first one-week seminar for physicians to be held at the institute from January 14 to 19, inclusive.

Instruction will be under the supervision of Dr. J. Ernest Ayre, director of the Institute, and its research staff. More than twenty outstanding local and visiting physicians and scientists will compose the faculty. The general course of instruction in cancer diagnosis and cytology will include lectures, demonstrations and symposia covering the various branches of medicine as related to cancer, including clinical, cytological, surgical and histopathological fields.

Interested physicians should direct their inquiries regarding qualifications, registration, fees and other details to the Director of the Dade County Cancer Institute at 1155 North West 14th Street, Miami, Florida. Applications for registration, limited to thirty-five physicians, will be accepted through January 12.

NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

The fifteenth annual meeting of the New Orleans Graduate Medical Assembly will be held March 10 to 13, with headquarters at the Municipal Auditorium in New Orleans.

Eighteen outstanding guest speakers will participate,

(Continued on page 1204)

it's TIME to consider . . .

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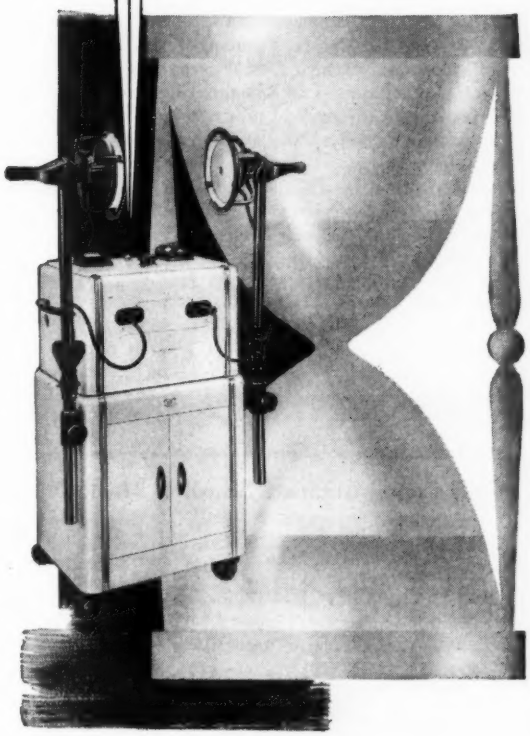

Yes, time is running out. F.C.C. restrictions on diathermy frequency and harmonics will be in full effect June 30, 1952.

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We'll be glad to send complete information. Naturally, there's no obligation . . . and we'll include the new chart "Modern Medical Diathermy Technics" showing 22 typical treatment applications in *full color*. Fill out the coupon and mail it now!

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NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

(Continued from Page 1202)

and their presentations will be of interest to both specialists and general practitioners. In addition, the program will include symposia on "Resuscitation" and "Complications of Antibiotic Therapy and Their Management," daily demonstrations of medical and surgical procedures in color television, clinicopathologic conferences, medical motion pictures, over 100 technical exhibits and three round-table luncheons.

The assembly has planned a postclinical tour to follow the 1952 meeting in New Orleans. On March 15 a party composed of doctors and their families will leave by plane for Merida, Yucatan, Mexico. The itinerary also includes Mexico City, Cuernavaca, Taxco and Acapulco in addition to other sightseeing. Arrangements have been made for medical programs and visits to hospitals in Merida City. The group will return to New Orleans on Saturday, March 29.

Details of the New Orleans meeting and the postclinical tour are available at the office of the assembly, Room 103, 1430 Tulane Avenue, New Orleans 12, Louisiana.

U. S. PUBLIC HEALTH SERVICE

A competitive examination for appointment as medical officer to the Regular Corps of the U. S. Public Health Service will be held on February 5, 6 and 7 at various points in the country. Applications must be received not later than January 2.

Cook County Graduate School of Medicine

ANNOUNCES CONTINUOUS COURSES

SURGERY—Intensive Course in Surgical Technic, two weeks, starting January 21, February 4, February 18.
Surgical Technic, Surgical Anatomy and Clinical Surgery, four weeks, starting March 3, June 2.
Surgical Anatomy and Clinical Surgery, two weeks, starting March 17, June 16.
Surgery of Colon and Rectum, one week, starting March 3, April 7.
Gallbladder Surgery, ten hours, starting April 21.
Basic Principles in General Surgery, two weeks, starting December 10, March 31.
Breast and Thyroid Surgery, one week, starting June 23.
Esophageal Surgery, one week, starting June 23.
Thoracic Surgery, one week, starting June 2.

GYNECOLOGY—Intensive Course, two weeks starting February 18, March 17.
Vaginal Approach to Pelvic Surgery, one week, starting March 3, March 31.

OBSTETRICS—Intensive Course, two weeks, starting March 3, March 31.

MEDICINE—Intensive General Course, two weeks, starting May 5.
Electrocardiography and Heart Disease, two weeks, starting March 17.
Gastroenterology, two weeks, starting May 19.
Hematology, one week, starting June 16.

UROLOGY—Intensive Course, two weeks, starting April 28.
Ten-day Practical Course in Cystoscopy, starting January 7, January 21, and every two weeks.

ROENTGENOLOGY—Two-week Lecture and Clinical Courses each month.

General, Intensive and Special Courses in All Branches of Medicine, Surgery and the Specialties

TEACHING FACULTY—ATTENDING STAFF OF COOK COUNTY HOSPITAL

ADDRESS: REGISTRAR, 427 South Honore Street Chicago 12, Illinois

Appointments will be made in the grades of assistant surgeon and senior assistant surgeon. Appointees to the former grade must have had at least seven years of education and professional experience since graduation from high school, while appointees to the latter grade must have had at least ten years.

Gross pay is the same as that for equivalent rank in the army—entrance pay for an assistant surgeon with dependents, $5,686, and for a senior assistant surgeon, $6,546.

Application forms may be obtained from the Surgeon General, U. S. Public Health Service, Federal Security Agency, Washington 25, D. C. Attention: Division of Commissioned Officers.

MINNESOTA SOCIETY OF NEUROLOGY AND PSYCHIATRY

The regular meeting of the Minnesota Society of Neurology and Psychiatry was held in Saint Paul on November 13. The scientific program was as follows: Dr. Fae Tichy, Minneapolis, appearing by invitation, presented a paper on "Barbiturate Encephalitis." Dr. Maurice Klotz, St. Cloud, presented his inaugural thesis on "Evaluation of Repeated Courses of ECT in Chronic Schizophrenia; Preliminary Report."

RHEUMATIC FEVER CONFERENCE

A three-day symposium on rheumatic fever was held at the University of Minnesota, November 29 to December 1, under the joint sponsorship of the University and the Minnesota Heart Association.

Lectures on various aspects of rheumatic fever were presented by leading specialists from throughout the country. Among the speakers was Dr. Morse J. Shapiro, Los Angeles, formerly of Minneapolis.

CONTINUATION COURSES

Electrocardiography.—The University of Minnesota will present a continuation course for general physicians in electrocardiography, January 21 to 25. Emphasis will be placed on practical experience in reading electrocardiograms. Each registrant will be given an opportunity to interpret approximately 200 tracings. These will later be reviewed and interpreted by the teaching staff. Illustrated lectures will supplement the practical sessions. The faculty for the course are members of the staff of the University of Minnesota Medical School and the Mayo Foundation.

Clinical Neurology.—Dr. Harold G. Wolff, professor of medicine and associate professor of psychiatry, Cornell University Medical College, New York City, will give the annual J. B. Johnston Lecture at 8:15 p.m. on January 30. Dr. Wolff's subject will be "On the Nature of Pain." He will also participate in a continuation course for physicians in clinical neurology which will be presented at the Center for Continuation Study from January 28 to February 9. Other visiting faculty members for the course include Dr. Benjamin Boshes, associate professor of neurology, Northwestern University Medical School, Evanston, Illinois; Dr. H. Houston Merritt, professor of neurology, Columbia University,

and director of the Service of Neurology, Neurological Institute, Presbyterian Hospital, New York City; and Dr. Henry G. Schwartz, professor of neurosurgery, Washington University School of Medicine, St. Louis, Missouri. Dr. A. B. Baker, professor and director of the Division of Neurology, is chairman for the course and will be joined by members of the faculty of the University of Minnesota Medical School and the Mayo Foundation.

E. STARR JUDD LECTURE

The nineteenth E. Starr Judd Lecture will be given by Dr. Thomas G. Orr, professor of surgery, University of Kansas, Kansas City, Kansas, on March 20 at 8:15 p.m., in the amphitheater, Owre Hall (formerly Medical Sciences), sponsored by the University of Minnesota Medical School. Dr. Orr's subject is "Some Observations on the Treatment of Carcinoma of the Pancreas."

The late E. Starr Judd, an alumnus of the Medical School of the University of Minnesota, established this annual lectureship in surgery a few years before his death.

LYON-LINCOLN COUNTY SOCIETY

At a meeting of the Lyon-Lincoln County Medical Society at Marshall on November 6, Dr. Kenneth A. Peterson, Marshall, was elected president of the organization. Dr. R. R. Remsberg, Tracy, was named vice president, and Dr. G. Harland Purves, Hendricks, secretary-treasurer.

Principal speaker at the meeting was Dr. H. A. Shaw, Minneapolis, who spoke on eye infections and treatment.

SOUTHWESTERN MINNESOTA MEDICAL ASSOCIATION

The Southwestern Minnesota Medical Association held its annual meeting at Jackson on October 29. Officers elected were Dr. S. S. Chunn, Pipestone, president; Dr. W. B. Wells, Jackson, president-elect, and Dr. O. M. Heiberg, Worthington, secretary. Guest speaker at the meeting was Dr. Tague C. Chisholm, Minneapolis, who discussed acute conditions of the abdomen in children.

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Woman's Auxiliary

LEGISLATIVE REPORT

Mrs. H. F. R. Plass, Legislative Chairman

The 82nd session of Congress adjourned October 20 and will not convene until January 8, 1952. Generally speaking, the public's health was not harmed by legislation enacted. However, many bills were introduced which will be discussed when Congress convenes in January and efforts must not be relaxed in expressing viewpoints to Congressmen.

S. 337 (The Federal Aid to Medical Schools), which was opposed by the AMA, was blocked for consideration this session by Senator Connolly. It will be reconsidered in January. Auxiliary members who haven't written their Congressmen, should do so now.

H.R. 3298—The Prescription Drug Bill passed in the form approved by the American Medical Association. The Federal Security Administration will *not* decide which drugs are prescription drugs.

The Jenner amendment to the tax bill made it legal for states to publish the names of people receiving public assistance. This has been a hotly contended issue. Oscar Ewing tried to withhold federal funds from the State of Indiana for publishing the names of people receiving aid. Mr. Ewing is not pleased with the Jenner amendment; he feels it is an invasion of privacy. Indiana, on the other hand, proved publication of the list cut down the amount of money needed for assistance programs and was instrumental in detecting fraudulent claims.

Besides the Federal Aid to Medical Schools bill, the following legislation has been introduced for January discussion. Though the American Medical Association has not taken a definite stand on much of it, it opposes the Federal Aid to Medical Schools and any form of compulsory health insurance.

1. S. 2171—Voluntary Health Insurance Bill introduced by Mr. Hill of Alabama and Mr. Aiken of Vermont. This bill is similar to the one introduced in the 81st Congress and the same dangers to the medical profession are inherent in this bill. It is interesting to note that Dr. Bauer, AMA President-elect, has again made a plea for voluntary plans. However, Dr. Bauer pointed out three defects in existing voluntary plans which we must strive to correct. These defects are:

(a) Need of individual insurance as well as group protection.

(b) Provision for people over sixty-five years of age.

(c) Protection against financially catastrophic illness.

2. S. 2337, introduced by Mr. Lehman of New York, would provide for state aid, through use of federal funds, for maternity and infant care for wives and infants, and hospital care for *all* dependents of enlisted members of the Armed forces during the present emergency.

3. S. 2246 introduced by Mr. Lehman would give more aid to the states to extend and improve health services for the prevention and reduction of chronic diseases.

4. S. 2247 introduced by Mr. Lehman would authorize grants to the States to extend and improve diagnostic out-patient health services.

5. S. 2248 introduced by Mr. Lehman would authorize loans to assist in the establishment of clinics or medical groups, designed to afford improved diagnostic services or improve diagnostic or curative services.

6. S. 2301 introduced by Mr. Ives of New York, provides a program of grants and scholarships in the field of nursing.

7. S. 2325 introduced by Mr. Humphrey is to provide and maintain safe and healthful conditions of employment in industries affecting commerce.

8. S.J. Res. 110 introduced by Mr. Murray would provide federal funds for study of the causes and effects of air pollution and its prevention and cure.

9. H.R. 5795 introduced by Mr. Mansfield of Montana would establish a National Multiple Sclerosis Institute within the Public Health Service.

10. H.R. 5853 introduced by Mr. Priest of Tennessee would provide federal funds allotted to state programs wherein health instruction, safety instruction, and physical education is provided in public elementary and secondary schools.

One last report. The Congress overrode the President's veto of a bill to provide free autos to veterans of the Korean war who had lost a leg or arm or are blind.

FALL CONFERENCE TERMED SUCCESS

Approximately eighty doctors' wives registered for the annual Fall Conference and School of Instruction, held October 12 at the Hotel Lowry. After the showing of the film "Here's Health—the American Way," the Auxiliary president, Mrs. F. P. Moersch, Rochester, Minnesota, called the board meeting to order at 10 a.m.

She presented greetings to auxiliary members representing county auxiliary organizations throughout the entire state. She then called for the reading of the Secretary's and Treasurer's reports.

The following committee reports were then presented:

Bulletin—Mrs. John Dordal, Sacred Heart, chairman, urged all members to subscribe, stating that, "an informed auxiliary will be an efficient auxiliary."

Cancer—Mrs. L. P. Howell, Rochester, chairman, presented a résumé of the work done by various national cancer organizations, including details of allocation of funds, urging members to help by (1) making cancer dressings, (2) helping transport older and disabled people to places where they receive their series of x-rays, (3) helping with "local loan closet"—scarce supplies stored and loaned, (4) visiting cancer patients, (5) having alert local chairman to work with county commanders; the doctor's wife can often provide the medical tie-in for moral support in the cancer drive, (6) working with

(Continued on Page 1208)

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(Continued from Page 1206)

their husbands, taking advantage of information seen in professional and lay films available.

Today's Health—Mrs. B. M. Black, Rochester, urged Auxiliary members to subscribe to *Today's Health* and appoint chairmen in each county.

Legislation—Mrs. L. Raymond Scherer, Minneapolis, for Mrs. H. F. R. Plass, Minneapolis, chairman, reported that the committee has sent legislative posters to all members, a letter to all county chairmen with outline of national legislation, legislation chairmen's names reported to the national auxiliary. Future plans include: monthly report in MINNESOTA MEDICINE, close contact with each county chairman on legislative matters. Chairman urged extensive use of the legislative card and advised that members should "never cease to be polite" in voicing opinions on legislation to Congressmen.

Program and Health Education—Mrs. David Halpern, Brewster, chairman, outlined four aims of this committee's work: (1) strive to make every doctor's wife well informed; (2) promote friendship among doctors' wives and families; (3) increase awareness of being informed and the importance of auxiliary work; (4) train each doctor's wife to participate in all local health programs and health projects.

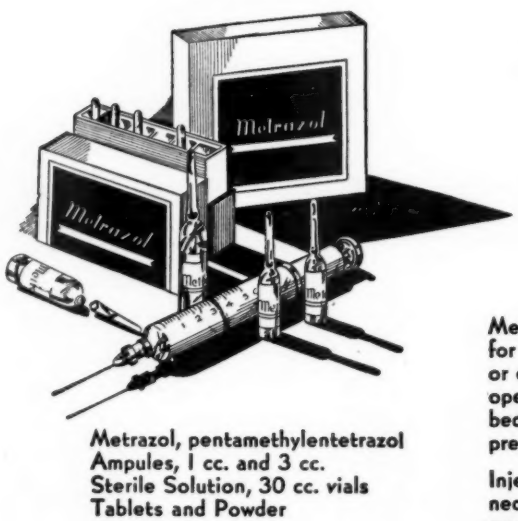
In advising how to organize a program, the chairman suggested the programs be short, interesting and varied, well organized to include possibly a lecture, group discussion, study course or films. Some time should be saved for a social hour. Use the auxiliary pledge. Consult the local medical society before taking on a new project.

Suggestions for program material include: (1) study the American Medical Association, its history, function, bureaus, councils or departments; (2) twelve-point program of the AMA to know what it supports; (3) health council study; (4) study voluntary health insurance plans in the state; (5) health problems in your community, adopt and promote projects; (6) medical legislation, nurse recruitment, *Today's Health*, civil defense; (7) material in *Bulletin*, auxiliary section of MINNESOTA MEDICINE, auxiliary *Newsletter;* (8) have publicity for program; (9) maintain a speaker's bureau; (10) sponsor radio and television programs of the AMA.

Public Relations—Mrs. L. J. Leonard, Minneapolis, chairman, stressed again the importance of being informed on the continuing dangers of socialized medicine. Members should be alert to answer intelligently the questions of lay people. Be aware of health needs in the community and join all community health projects, work closely with the newspapers.

Press and Publicity—Mrs. A. B. Rosenfield, Minneapolis, chairman, read a resolution passed by the press women's organization against socialized medicine.

Health Days—Mrs. W. G. Johanson, St. Paul, chairman, told auxiliary members that the purpose of health days is to bring health information to the community. Health Days are well worth the effort—the follow-up value is great, for it brings an appreciation to the community of the doctor and his work. Health Days can help give an over-all undergirding of health and well being of the people of Minnesota.



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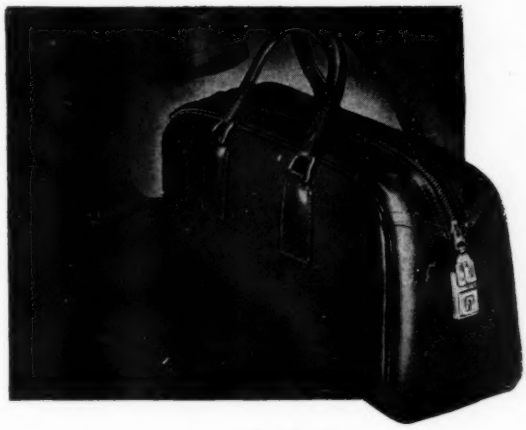

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Medical and Surgical Relief—Mrs. W. P. Ritchie, St. Paul, for Mrs. L. G. Culver, St. Paul, chairman, urged counties to appoint committees to collect surplus supplies that accumulate in doctors' offices and help get them to places where shortages exist.

Organization—Mrs. C. L. Sheedy, Austin, president-elect and chairman, reported the organization of Steele County Medical Auxiliary, also reported that thirty out of thirty-two component medical societies now have auxiliaries. "We need the auxiliary and the auxiliary needs us. Not only be organized, but be active."

Emergency Nursing—Mrs. Ivar Sivertsen, Minneapolis, chairman, reported that in 1950 Minnesota's nurse requirement of 1,215 was nearly filled; 1,211 girls were secured for nurses' training. The University of Minnesota has taken on a larger class. The Auxiliary was urged to co-operate with the local district nurse's enrollment chairman and to spread the message of the need for nurses to local girls.

Archives—Mrs. J. M. Neal, St. Paul, chairman, reported that the aim of the Archives Committee is to complete a history file for each county medical auxiliary.

The president then called for nominations for membership on the Nominating Committee. Members chosen were: Mrs. Harold Benjamin, Minneapolis, chairman; Mrs. C. W. Froats, St. Paul; Mrs. H. E. Bakkila, Duluth; Mrs. P. A. Lommen, Austin; Mrs. M. I. Hauge, Clarkfield.

Mrs. Charles W. Waas, immediate past president, voiced her thanks to last year's committee chairmen, officers, regional advisors, all board members, and especially to the Olmsted-Houston-Fillmore-Dodge County Medical Auxiliary for the fine annual meeting in Rochester.

It was decided to hold the midwinter board meeting at the Curtis Hotel, Minneapolis, Wednesday, January 16, 1952. Please note the change in date from that given in the *Newsletter*.

The meeting was adjourned at approximately 12:30 p.m.

Average length of life in the United States has increased to a record high of nearly sixty-eight years, according to Federal Security Administrator Oscar R. Ewing.

The new figure, based on final 1949 vital statistics compiled by the Public Health Service, shows a gain of almost half a year over the average lifetime indicated by 1948 death rates.

White women, on the average, live longer than any other group, outliving white men by more than five years. The average lifetime expected for white women at birth is seventy-one and one-half years, while the average for white men is sixty-five years, eleven months.

Negroes and other non-white groups have a shorter average life—fifty-eight and one-half years for non-white men, and sixty-two years eleven months for non-white women. Although white persons live longer than non-white, the difference has been sharply reduced. In 1900, whites outlived non-whites by an average of about fifteen years, compared to about eight years in 1949.

While the expectation of life at birth has increased by more than twenty years since the turn of the century, this has been almost entirely due to prolonging the lives of persons who formerly would have died in infancy, childhood or young adulthood. This has resulted largely from the control of infectious diseases.

In Memoriam

ALFRED WASHINGTON ADSON

Dr. Alfred W. Adson, chief of the Section on Neurosurgery at the Mayo Clinic until 1946 when he became senior consultant, died on the morning of November 12, 1951, following a heart attack sustained while attending a meeting in Minneapolis the day before.

Dr. Adson was born in Terrill, Iowa, on March 13, 1887. He was born on a farm and got his preliminary education at Terrill before obtaining a B.Sc. degree from the University of Nebraska in 1912. After finishing two years at the medical school of the University of Nebraska, he took the last two years at the University of Pennsylvania medical school, acquiring his M.D. degree in 1914. He immediately came to the Mayo Clinic, on July 1, 1914, as a Fellow in Surgery.

Dr. Adson was assisting Dr. Emil Beckman who at that time was doing the brain surgery of the clinic. When Dr. Beckman died suddenly in 1916, Dr. Adson took over on short notice and operated on the patients scheduled for operations. It has been stated that Sir Berkley Moynihan, the celebrated surgeon of Leeds, England, when asked what during his visit to the United States had impressed him most, replied that seeing a high school boy at the Mayo Clinic doing brain surgery was outstanding.

Dr. Adson was one of the founding members of the Society of Neurological Surgeons. Last September, fifty-nine of seventy-three former Fellows in Neurosurgery, who had been trained under Dr. Adson in the Mayo Clinic and Foundation, met for a reunion at Rochester in honor of their teacher and presented him with a map of the world showing the present locations of his former students—an impressive demonstration of the world-wide influence of this one teacher.

Dr. Adson was one of the first to use sympathectomy in the treatment of vascular diseases and hypertension on any great scale.

Dr. Adson was appointed to the State Board of Medical Examiners in 1929, was repeatedly reappointed, and was a member at the time of his death. He was several times president of the board.

He was president of the Minnesota State Medical Association in 1937 and was awarded the distinguished service medal of the State Association in July, 1947.

Dr. Adson took a great interest in medical organizational affairs and was active in the National Conference on Medical Services which meets yearly in Chicago. He, perhaps more than any other, was responsible for the establishment of an AMA office in Washington. He was also active in the North Central Conference, which was initiated in the thirties in the interests of better public relations, and served one year as president.

It was Dr. Adson who was responsible, with others, for instituting necessary legislation and establishing the Blue Shield in Minnesota.

Dr. Adson was well known nationally. He was seriously considered last June for the office of president-elect of the AMA and would doubtless have been in the running next June had not death overtaken him.

He served two three-year terms on the AMA Council on Medical Service and was chairman of the Council's seven-member Committee on Prepayment Hospital and Medical Service.

Not only was Dr. Adson an able surgeon, he was a national leader of men. He had ability as a speaker and could array and express his thoughts in logical sequence. His record was a good example of the strenuous life. He applied all his energy to anything he attempted, whether at work or play.

Dr. Adson is survived by his wife, Lora Smith; a daughter, Mrs. W. E. Wellman of Rochester, and a son, Lt. Martin Adson. Another son, William, died in World War II.

CHARLES F. ANDERSON

Mr. Charles F. Anderson, well known to physicians of Minnesota and the Twin Cities especially, suffered a heart attack in the Lowry Medical Arts building in Saint Paul about 6 p.m. on November 16, 1951, and died soon thereafter.

Charlie Anderson, president of the C. F. Anderson Co., Inc. of Minneapolis, had been in the medical supplies field most of his sixty-nine years, having been with Noyes Brothers and Cutler before he founded his own firm in 1919. His cheery disposition and willingness to accommodate gave him entré to physicians' offices everywhere, and he will be greatly missed.

A sister, Mrs. Fred Johnson, died less than twenty-four hours before in a fire which partially destroyed her home in Saint Paul.

He is survived by his widow, a son David of Minneapolis, and two sisters, Hilda and Mrs. Florence Raschke, both of Saint Paul.

JAMES BLAKE

Dr. James Blake, for nearly fifty years a general practioner at Hopkins, Minnesota, died October 28, 1951, at the age of eighty-five.

Dr. Blake was born in Ontario April 2, 1866. After completing grade school at the age of sixteen, he came to the states with the idea in the back of his mind of becoming a doctor. Not satisfied with available jobs in Chicago, he rode a freight train to Minnesota and obtained a job as a farm hand at Graceville, Minnesota. After spending three years in saw mills of Wisconsin and Michigan, he returned to Minnesota and got a job in a pulp mill at Little Falls. Having saved some money, he enrolled at Mankato normal school and completed the four years' course in two years. This encouraged him to take up the study of medicine at the University of

(Continued on Page 1212)

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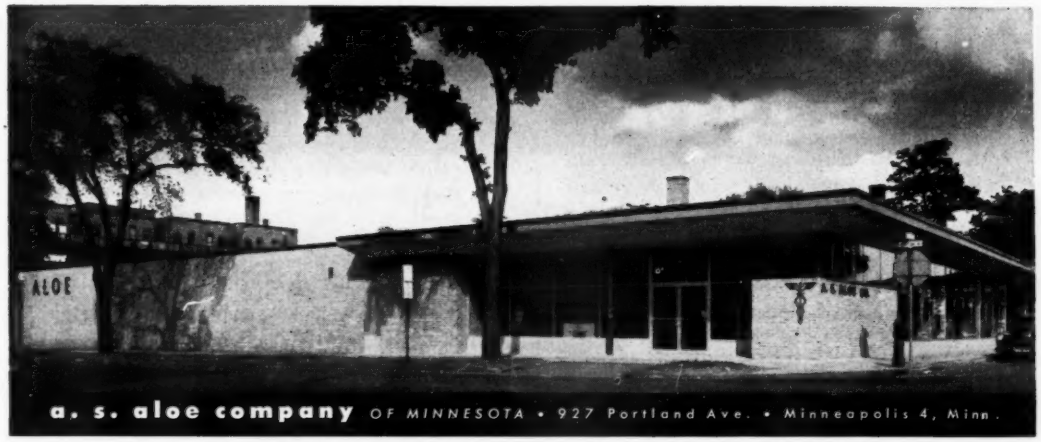

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Gastroenterology
Ralph A. Reis, M.D., Chicago, Ill.
Gynecology
Charles A. Doan, M.D., Columbus, Ohio
Medicine
John P. Merrill, M.D., Boston, Mass.
Medicine
William Parson, M.D., Charlottesville, Va.
Medicine
Jacob E. Finesinger, M.D., Baltimore, Md.
Neuropsychiatry
Leroy A. Calkins, M.D., Kansas City, Kan.
Obstetrics
John H. Dunnington, M.D., New York, N. Y.
Ophthalmology
Edward L. Compere, M.D., Chicago, Ill.
Orthopedic Surgery
Hayes Martin, M.D., New York, N. Y.
Otolaryngology
Oscar B. Hunter, M.D., Washington, D. C.
Pathology
Margaret M. Nicholson, M.D., Washington, D. C.
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Paul C. Swenson, M.D., Philadelphia, Pa.
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JAMES BLAKE

(Continued from Page 1210)

Minnesota where he graduated in 1901. After interning at Minneapolis General Hospital, he began practice at Hopkins in 1902.

His early practice included emergency surgery, such as appendectomies performed in home kitchens. He estimated that he had assisted in bringing some 5,000 babies into the world, for the most part home deliveries.

Dr. Blake built a clinic building at Hopkins in 1929. His two sons, Drs. Allen and James A. Blake, have been associated with him in recent years. A third son, Paul, is also a physician, and a fourth son, Donald, was killed in army maneuvers in California in World War II. One daughter, Mrs. Charles Adams of Springfield, Illinois, became a trained nurse, and Jeannette of Rapid City, South Dakota, studied to be a medical technician.

Mrs. Blake, in spite of her large family, was very active in the Woman's Auxiliary of the State and American Medical Associations, having served as president, first of the Minnesota State Auxiliary and later of the AMA Auxiliary.

Mrs. Blake died in 1948. In the fall of 1948, a testimonial dinner was tendered Dr. Blake by his many friends and acquiantances and was sponsored by the Veterans of Foreign Wars. Some 200 people attended.

A member of the Hennepin County Medical Society for years, Dr. Blake became a life member of the Minnesota State Medical Association in 1949.

The residents of Hopkins have lost one of its leading citizens in the death of Dr. Blake. His jovial nature and ready wit and, above all, his kindly disposition endeared him to his many friends.

JAMES FRANK CORBETT

Dr. James Frank Corbett of Minneapolis died suddenly November 6, 1951, at the age of seventy-nine.

Dr. Corbett was born at Chippewa Falls, Wisconsin, February 16, 1872. He attended Minneapolis schools and obtained his medical degree from the University of Minnesota in 1896. After interning at Minneapolis City Hospital, he took postgraduate work at Harvard and the University of Michigan.

He began private practice in Minneapolis in 1898 and at the same time was city bacteriologist. In 1907, he became assistant professor of surgical pathology at the University of Minnesota medical school.

Dr. Corbett served as a Lieutenant Colonel in the Medical Corps in World War I and was chief of neurological surgery at the General Hospital at Cape May, New Jersey. He was also chief of surgery at Walter Reed Hospital in Washington.

He was president of Hennepin County Medical Society in 1920 and of the Minnesota State Medical Association in 1923. He was a licentiate of the American Board of Surgery, a member of the American College of Surgeons, the Western Surgical Society, the American Society of Pharmacologists, the Minnesota Neurological Society and the Minnesota Academy of Medicine.

Dr. Corbett has no immediate survivors.

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Of General Interest

The Rockefeller Foundation, in its report for 1950 made public in September, showed that its appropriations amounted to $11,247,964. Of this amount, $2,326,840 was used for public health; $1,240,900 for medical sciences, and the balance for natural and social sciences, the humanities, scientific services and miscellaneous projects. Of the total, 65 per cent was for activities in the United States and 35 per cent in other countries.

* * *

Dr. Sam M. Mackoff has opened an office at 869 Lowry Medical Arts Building, Saint Paul, for the practice of allergy and diseases of the skin.

* * *

Dr. A. F. Giesen, Starbuck, attended the annual clinical congress of the American College of Surgeons at San Francisco during the first week of November.

* * *

A law has been passed by Congress entitling veterans serving on or after June 27, 1950, to the benefits of medical and hospital care, compensation and pensions which were provided for veterans of World War II.

* * *

Dr. Marcus A. Keil, formerly of Mankato, has opened an office at 327 Hyde Building, Albert Lea, for the practice of internal medicine.

* * *

The American Board of Obstetrics and Gynecology announces the election of **Dr. John L. Parks,** Washington, D. C., as a member and director of the board. Dr. Parks succeeds **Dr. Joseph L. Baer,** who has been vice president of the board for over twenty years and who has resigned.

* * *

Leaders from twelve Minnesota communities met at the University of Minnesota, November 15 to 17, to discuss **mental health.** Communities represented were Albert Lea, Anoka, Cloquet, Crookston, Fergus Falls, Little Falls, Mankato, Pelican Rapids, Preston, St. Cloud, Stillwater and Willmar.

First of its kind ever conducted in the state, the conference was attended by lawyers, judges, doctors, nurses, clergymen, social workers and educators from the twelve towns. At the meeting these community leaders were shown how they could co-operate in tackling community mental health problems. Among the speakers, which included psychiatric social workers and mental health consultants, was **Dr. Richard M. Magraw,** neuropsychiatry instructor at the University of Minnesota.

The conference was sponsored by the University, Minnesota State Medical Association, Minnesota Probate Judges' Association and the state departments of education, health and social welfare.

At the annual meeting of the American College of Ophthalmology and Otolaryngology, held at the Palmer House, Chicago, on October 18, **Dr. William L. Benedict,** Rochester, was re-elected executive secretary-treasurer; **Dr. Lawrence R. Bois,** Minneapolis, secretary for home study courses, and **Dr. Erling W. Hansen,** Minneapolis, secretary for public relations.

* * *

Dr. Anthony J. Lund, formerly of Bismarck, North Dakota, has joined the staff of the Starekow Clinic in Thief River Falls. A graduate of the University of Illinois School of Medicine, Dr. Lund served his internship at Ancker Hospital, Saint Paul.

* * *

Dr. Merton A. Johnston has moved from Storden to Nevada, Iowa, where he has taken over the Houston Clinic. Dr. Johnson began practice at Storden shortly after World War II.

* * *

Principal speaker at the annual meeting of the Blue Earth County Public Health Association in Mankato on October 17 was **Dr. Robert N. Barr** of the Minnesota Department of Health. In his talk Dr. Barr pointed out that two of the biggest problems in public health today are the care of the aged and the prevention of accidents.

* * *

Dr. Hendrick J. Svien, Rochester, was elected president of the Congress of Neurological Surgeons at a meeting in Memphis, Tennessee, on November 15.

* * *

Among those attending the thirty-seventh annual clinical congress of the American College of Surgeons at San Francisco during the first week of November was **Dr. William A. Black** of New Ulm.

* * *

Dr. Leo J. Rigler, head of the department of radiology and physical medicine at the University of Minnesota, recently returned from a two-month tour of Israel and Iran. He made the trip as a member of a fourteen-man medical mission sent out by the World Health Organization and the Unitarian service committee to explain the latest advances in treating patients and teaching medical students.

Dr. Rigler reported that there is a tremendous contrast between Iran and Israel, including their respective physician-population ratios. Teheran, capital of Iran, has 1,000 physicians for its 1,000,000 population, a ratio about equal to most European cities. The rest of Iran has only 1,500 physicians for a population of more than 15,000,000—one of the lowest ratios in the world.

Israel, on the other hand, has the highest percentage of physicians in the world. However, it

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1951	12,000	250.00	5%	130.00	2.60%

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is facing a future medical problem, since most of its doctors, refugees from Europe, are over fifty years of age. Israel's medical school, which just graduated its first class (fifty-two doctors), must find a way to supply enough new physicians for the future.

In Iran, Teheran's new medical school is now graduating 200 physicians a year. Iran is also organizing five new schools and hopes to turn out soon 1,000 doctors a year to care for its poorly supplied population.

* * *

After three years of practice in Gibbon, **Dr. John H. Glaeser** has closed his office and moved to Minneapolis. He is now taking a three-year course of study in ophthalmology and otolaryngology at the University of Minnesota.

* * *

Dr. Gaylord W. Anderson, director of the University of Minnesota School of Public Health, became president of the American Public Health Association at the organization's meeting in San Francisco on November 1.

* * *

Dr. G. M. A. Fortier, Little Falls, received notice on October 18 that he had been accepted into the International Academy of Proctology.

* * *

A second heart research grant for 1951 has been

made to the University of Minnesota by the Minnesota Heart Association. Early in November, **Dr. Thomas J. Dry,** Rochester, chairman of the group's allocations committee, sent a check for $20,000 to **Dr. Harold S. Diehl,** dean of medical sciences at the University. A previous research grant of $25,000 had been made in August.

* * *

Dr. L. M. Eaton, Rochester, was elected vice president of the Central Neuropsychiatric Association at its twenty-seventh annual convention in Minneapolis on October 19 and 20. Dr. Eaton, professor of neurology in the Mayo Foundation, had served as secretary-treasurer of the organization during the previous year.

* * *

At the annual North Central Medical Conference, held November 11 at the Hotel Radisson, Minneapolis, **Dr. Floyd Rogers,** Lincoln, Nebraska, was elected president; **Dr. H. Russell Brown,** Watertown, South Dakota, president-elect, and **R. R. Rosell,** Saint Paul, secretary-treasurer. The meeting was attended by approximately 100 physicians and medical society executives from Wisconsin, North Dakota, South Dakota, Nebraska, Iowa and Minnesota. An unusually stimulating program was presented, with emphasis on the human relations element in public relations.

* * *

Professor C. H. Andrewes, National Institute for Medical Research, University of Leeds, London, England, will give the annual George Chase Christian Cancer Lecture at the University of Minnesota Medical School on the evening of January 15. His subject will be "Current Thoughts on Viruses and Cancer."

* * *

A $7,500 annual grant to the University of Minnesota for tuberculosis research was approved at the annual meeting of the Minnesota Public Health Association on October 22. The fund will be known as the H. Longstreet Taylor grant and is named after the late Dr. Taylor, who was active in the state campaign for prevention of tuberculosis. The grant will be administered by **Dr. J. Arthur Myers,** professor of preventive medicine and public health at the University.

* * *

Dr. Lydia Neibergs has announced her association in the practice of ophthalmology with **Dr. Karl C. Wold,** 1051 Lowry Medical Arts Building, Saint Paul. She is a graduate of the University of Latvia at Riga, where she also took her postgraduate work. Dr. Neibergs has practiced ophthalmology for thirteen years in Latvia and Germany.

* * *

Dr. Joseph E. Christopherson, a former resident of Virginia, now practicing at Mason City, Iowa, was recently certified in the specialty of general surgery by the American Board of Surgery.

* * *

Dr. Albert V. Stoesser, Minneapolis, has been ap-

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pointed a member of the Subcommittee on Allergy of the American Board of Pediatrics.

* * *

Dr. C. W. Rogers, Winona, has been notified of his election as a fellow of the American Academy of Pediatrics.

* * *

At a reorganization meeting of the Virginia Health Council on October 22, **Dr. W. S. Neff,** Virginia, was reappointed chairman of the council. Discussions were held on two proposed projects for the year: the possible establishment of an alcoholic rehabilitation center at Virginia and a vision survey.

* * *

Principal speaker at a meeting of the Red Wing Society for the Mentally Retarded, at Red Wing on October 23, was **Dr. E. J. Engberg,** superintendent of the Faribault Colony for the mentally retarded. Dr. Engberg described the steps taken on admission of a patient to the Faribault Colony to determine the correct program of care to be followed.

* * *

Two natives of Rochester, both of whom completed fellowships at the Mayo Clinic in October, have moved to other cities to practice. **Dr. Robert U. Moersch,** son of Dr. and Mrs. F. P. Moersch, Rochester, is practicing surgery at Clearwater, Florida. **Dr. J. L. Crenshaw, Jr.,** son of Dr. and Mrs. J. L. Crenshaw, Rochester, has moved to Grand Junction, Colorado. He was a fellow in obstetrics and gynecology.

Among the speakers at a mental health meeting for the public at Coleraine on October 18 was **Dr. Henry Hutchinson,** superintendent of the Moose Lake State Hospital. Following a discussion of the common mental ailments and their causes, he conducted a question-and-answer session on the subject.

* * *

The election of four Minneapolis physicians as fellows of the American Academy of Pediatrics was announced on November 7. The four are **Dr. Charles Upton Lowe, Dr. Lloyd Stanley Nelson, Dr. Sidney S. Scherling** and **Dr. Theodore S. Smith.**

* * *

Dr. Norval Westby, Madison, was guest speaker at a meeting of the Madison Business Women's Club on October 29. Dr. Westby discussed some of the more recently developed drugs and described their purposes and uses.

* * *

Dr. Edward L. Tuohy, Duluth, has been elected to a three-year term as trustee of the Greater University Fund, the fund-raising project of the University of Minnesota.

* * *

Dr. Louis L. Flynn, Saint Paul, pediatric psychiatrist, participated in a discussion on "Emotional Health of Our Child" at a meeting of the Minnesota Congress of Parents and Teachers at Saint Paul on October 25. Dr. Flynn talked about the role of parents in children's behavior.

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Dr. P. H. Rockwood, of the Fergus Falls Clinic, attended a postgraduate course in obstetrics and gynecology sponsored by the Chicago Medical Society and held at Northwestern University Medical School from October 22 through 26.

* * *

Fifty-three new fellows in the Mayo Foundation were accepted for fellowship work beginning with the October quarter, it was announced on October 17. Fourteen of the physicians are from other countries.

* * *

Among the honored guests at a thirtieth anniversary luncheon of the Faribault Lions Club on October 18 was **Dr. C. M. Robilliard,** Faribault, a charter member of the organization.

* * *

The fourth annual convention of the Minnesota Association of Nursing Homes was held at Rochester on October 23 and 24, with the principal speakers consisting of **Dr. Jorge A. Lazarte,** of the Rochester State Hospital, and **Dr. N. O. Hanson,** of the Mayo Clinic. Dr. Lazarte spoke at the annual banquet, while Dr. Hanson presented a talk on "Care of the Aged."

* * *

Dr. Owen H. Wangensteen, chairman of the department of surgery at the University of Minnesota, has written a monograph on cancer of the esophagus and stomach to be sent to 190,000 physicians, senior medical students and teachers.

The 112-page booklet is the sixth in a series on early recognition of cancer published by the American Cancer Society. In it are emphasized the practices followed at the University Hospitals, especially in regard to patients who have been operated upon after cancer has spread to lymph nodes.

From November 5 to 9 Dr. Wangensteen, with eight of his University colleagues, attended the thirty-seventh clinical congress of the American College of Surgeons at San Francisco. At the meeting Dr. Wangensteen conducted a daily forum on fundamental surgical problems, designed to bring out new and experimental work in surgery.

* * *

Mantoux tests were given to pupils in the first, fifth, ninth and twelfth grades in all village schools and to all Indian children and school faculty members and employes in Mahnomen County on October 22. Giving the tests were **Dr. E. A. Leggett,** of the Minnesota Department of Health, and **Dr. K. W. Covey** and **Dr. K. A. Danford,** both of Mahnomen.

It is planned to conduct the testing program each year, thereby keeping a check on children periodically during their school career.

* * *

Two brothers, both graduates of the University of Minnesota Medical School and former residents of Rochester, have opened a new medical and surgical

clinic at Lake Crystal. They are **Dr. Paul M. Smith,** who has practiced at Lake Crystal since the end of World War II, and **Dr. Harry J. Smith,** who recently completed his internship at Milwaukee General Hospital.

* * *

The twenty-seventh annual reunion of the Alumni Association of the Mayo Foundation was held in Rochester on October 18, 19 and 20, with a total registration of 182 physicians in attendance.

The program for the three-day meeting included surgical clinics, seminars, scientific sessions, award ceremonies and election of new officers.

Co-recipients of awards for meritorious research were **Dr. M. A. Block** and **Dr. F. Henry Ellis,** both fifth-year fellows in surgery, and **Dr. T. B. Fitzpatrick,** now a member of the faculty of the University of Michigan Medical School. A special award of appreciation went to Miss Isabel Farr, Rochester, who recently retired as secretary of the Mayo Foundation after serving in the Foundation office since it opened thirty-six years ago.

The fifth Judd-Plummer Memorial Lecture was delivered, following the award ceremonies, by **Dr. Shields Warren,** professor of pathology at Harvard Medical School and director of the division of biology and medicine of the Atomic Energy Commission. Dr. Warren spoke on "The Biologic Effects of Atomic Radiation."

Officers of the association for the coming year were elected as follows: **Dr. Donald C. Balfour,** honorary president; **Dr. John L. Kleinheksel,** Witchita, Kansas, president; **Dr. Nat H. Copenhaver,** Bristol, Tennessee, first vic president; **Dr. Walter C. MacKenzie,** Edmonton, Canada, second vice president; **Dr. James F. Weir,** Rochester, secretary, and **Dr. David G. Pugh,** Rochester, associate secretary-treasurer.

General chairman for the reunion this year was **Dr. Haddon M. Carryer,** Rochester.

* * *

Dr. A. H. Borgerson, Long Prairie, became a fellow in the International College of Surgeons and was certified in general surgery by the international board of the College at a meeting of the Canadian and United States chapters at Chicago in October.

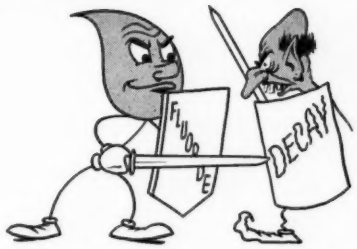
HOSPITAL NEWS

A ground-breaking ceremony on October 28 started construction work on the new $1,050,000 **Bethesda Hospital** to be erected at Crookston. The three-story hospital, which will take about one year to complete, will have a maximum capacity of seventy-seven beds. A fourth floor could be added in the future to house an additional fifty beds.

Speakers at the ground-breaking ceremony included Dr. M. O. Oppegaard, mayor of Crookston; Walter J. Ross, Fisher, president of the hospital board of directors; Dr. J. F. Norman, Crookston, president of the Minnesota State Medical Association, and Dr. D. E. Pohl, director of the hospital medical staff.

When the new hospital is completed, the present

Important News!



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Bethesda Hospital in Crookston will be converted into a rest home.

The new $600,000 **Stevens County Memorial Hospital** was dedicated at Morris on November 18, and the first patients were moved into the fifty-bed hospital the following week.

The dedication ceremonies included talks by medical and hospital representatives and were followed by an open house at the hospital.

* * *

At a meeting of the **Roseau County** Lutheran Benevolent Association on October 22, Dr. D. O. Berge, chairman of the board of trustees, announced that construction of a proposed rest home was expected to start in the spring. He stated that to date the association had received $48 299, with $3,000 in additional pledges, to aid in financing the construction of the building. The association also planned to conduct a fund-raising campaign in the spring.

* * *

At a community dinner in Thief River Falls on October 24, before starting a fund-raising campaign for construction of a $250,000 **Northwestern Hospital** for the area, it was announced that gifts and pledges from the physicians of the medical staff and the hospital's board of directors already totaled more than $67,000.

Dr. Oscar F. Melby, Thief River Falls, honorary chairman of the building fund campaign, stated that the medical staff had pledged a total of $43,500. The board of directors had pledged $23,540.

Principal speaker at the meeting was Dr. Charles A. Haney, hospital advisor from Boston, Massachusetts.

* * *

A tour of the **Lakeside Memorial Hospital** at Pine City was a feature of a meeting of the Pine City Wednesday Study Club during the middle of October. During the tour Dr. E. G. Nethercott discussed the occupational therapy which he has encouraged among the hospital patients. Several patients displayed the products of their handicraft.

* * *

Dedication ceremonies for the new **St. John's Hospital** at Red Lake Falls were held on October 21. Among those who spoke at the dedication were Dr. L. N. Dale, Red Lake Falls, and Dr. J. F. Norman, Crookston.

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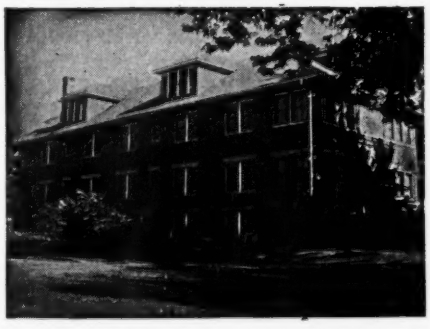
Minnesota Blue Shield as of September 30, 1951, attained an enrollment of 508,966 subscribers, a record of over half-a-million participants in the first four years of Blue Shield's operation in Minnesota. This achievement is outstanding and indicates the widespread desire for and interest in voluntary prepaid medical care plans. On a national basis Blue Shield enrollment reached twenty-one million subscribers, further testimony to the public's awareness of the need for this type of protection.

The increase in claims processed each month by Minnesota Blue Shield is also noteworthy. A total of 10,348 claims were processed during October compared to 6,009 claims in October, 1950. The October payment of $323,208.51 raises the amount paid for 1951 to $2,567,011, or more than twice the amount paid during the same period of 1950. These figures not only affirm the larger numbers of subscribers now receiving benefits, but also indicate an increasing utilization and gives Blue Shield an opportunity to prove its effectiveness in providing benefits at a time they are most needed.

During the fourth non-group Blue Cross and Blue Shield campaign which was held from October 21 through November 17, approximately 15,500 inquiries were received in the Blue Cross-Blue Shield office. Inquiries came from coupons appearing in advertisements in the daily papers, from radio spot announcements and from general correspondence.

Enrollment in Minnesota Hospital Service Association as of September 30, 1951, was 1,021,938. Payment to hospitals for the month of September totalled $809,198, bringing the total amount paid for 1951 to $8,417,185.

Mr. Calvin greeting the 500,000th Minnesota Blue Shield subscriber and the 21st millionth Blue Shield subscriber. Pictured left to right: Mr. James R. Wallace, Northern Ordnance plant worker, and the 500,000th Minnesota Blue Shield subscriber; Miss Rosemarie Hart, Northwest Airlines stewardess and the 21st millionth Blue Shield subscriber in the nation, and Mr. Arthur M. Calvin, Executive Director of the Minnesota Blue Cross-Blue Shield Plans.



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BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

ALLERGY IN RELATION TO PEDIATRICS. Bret Ratner, M.D., Professor of Clinical Pediatrics (Allergy) and Associate Professor of Immunology, New York Medical College; Attending Pediatrician, Flower and Fifth Avenue Hospitals; Director of Pediatrics, Sea View Hospital. Panel discussion. An official publication of The American College of Allergists. 228 pages. Price $3.75. Saint Paul: Bruce Publishing Co., 1951.

For many years, there has been a need for a text to appear dealing specifically with allergies in children. Within recent months two such books have been published—Pediatric Allergy by R. Chobot and Allergy in Relation to Pediatrics edited by Dr. Bret Ratner.

This latter book consists of a collection of papers written by some of the outstanding pediatric allergists in this country, seventeen contributors all told. The book begins with chapters on the genesis of allergy, formation of antibodies, and the relationship of infections to allergies. Following these chapters, there is a very good discussion of the diagnostic tests and their significance. The major allergic conditions—asthma, hay fever, and eczema, as well as some of the less commonly recognized allergies—neurological, gastro-intestinal, et cetera, are then taken up and their treatment outlined. A brief chapter on emotional problems in allergic children and a chapter on the practical office management of allergies in children complete the book.

As with any book consisting of a number of contributing authors, there is bound to be some variance in the quality of the contributions. Most of the chapters in this book are very well written and represent the summation of many years of experience in handling pediatric allergic problems. Other chapters, although also representing years of experience, are not written with the same critical scientific acumen.

This book is excellent and can be highly recommended. There is still need, however, for a more comprehensive book on pediatric allergies that could be used by the pediatrician and general practitioner as a reference book.

SHELDON C. SIEGEL, M.D.

METHODS IN MEDICINE; THE MANUAL OF THE MEDICAL SERVICE OF GEORGE DOCK, M.D., SC.D.: A COMPREHENSIVE OUTLINE FOR CLINICAL INVESTIGATION, MANAGEMENT, AND TREATMENT OF PATIENTS WITH VARIOUS MEDICAL DISORDERS. By George R. Herrmann, M.D., Ph.D., Professor of Medicine, University of Texas Medical Branch at Galveston; Director of the Cardiovascular Service and Heart Station, University of Texas Hospitals; Consultant in Medicine to the Surgeon General, U. S. Army; Consultant in Vascular Diseases to the Marine Hospital, U.S.P.H.S. 488 pages. Price $7.50. 2nd ed. St. Louis: C. V. Mosby Co., 1950.

Although Dr. Herrmann modestly describes this well-written manual as a "practical ward or bedside guide," it is far more comprehensive than one would expect from that statement. Well organized, it is outstanding because it clearly describes each procedure in adequate detail, so as to obviate the necessity for referral to other textbooks and journals.

Part III, Methods of Clinical Investigation, is particularly useful since it describes for each group of disorders such as allergies, heart disease, and endocrine disorders, the special studies valuable in that field. Part IV, Therapeutic Methods, presents modern, rational procedure and is so complete that it even contains the proprietary names and descriptions of the drugs recommended and discusses their relative value. The anxious reader is never left in midair by vague statements or generalities. The author gives him exact dosages, details of administration, results to be anticipated, et cetera. This manual is definitely *not* a condensation, digest, or epitome but, rather, a complete, useful, and valuable companion most physicians will want to own.

ROBERT J. BROTCHNER, M.D.

No one can be sure when the primary tubercle is going to resolve into a harmless primary complex, though most of them do. Only time will tell whether persons with scattered, irregular, and perhaps somewhat loose calcifications will remain clinically well, though the majority of them do. A tuberculous infiltration is always of interest on the basis of health protection and is of clinical importance unless it has reached the favorable discrete character described above.—GROVER C. BELLINGER, M.D., *Bulletin National Tuberculosis Association*, April, 1951.

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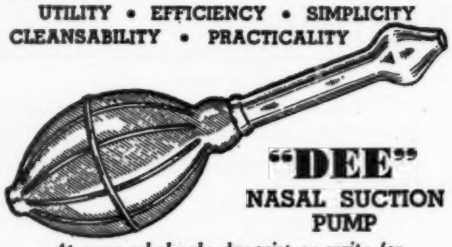

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BUY DEFENSE BONDS

MODIFIED AMPUTATION

(Continued from Page 1200)

cluded in the total tabulation since an estimate of healing was of course not possible.

In 65 per cent of the cases, primary or prompt healing was obtained. In only 17 per cent of the cases was revision necessary to obtain a satisfactory stump. The classification of prompt healing was applied to those cases in which there was healing of at least part of the wound by secondary intention. The term "delayed healing" was applied to those cases in which there was drainage for prolonged periods and finally healing by granulation and epithelialization but in which revision was not required to achieve a satisfactory stump.

There has been no attempt made to review the mortality figures in this presentation. It is not felt that the amputation *per se* has added significantly to mortality in these extremely ill patients. More often the amputation has been a life-saving measure. Although a few of the patients presented finally succumbed to their disease while still in the hospital following operation, a satisfactory time for the evaluation of the amputation and the resulting stump had elapsed.

Discussion

DR. E. EVANS: I haven't too much to say, but I have some material that might be of some help. Several of you men who were in the Service know the guillotine amputations were left open not because that was good operative technique, but because in the subsequent transportation of those patients wounds must be left open so that no subsequent infection can result. By leaving them open, good drainage was accomplished.

The rule in the Army was open, and guillotine procedure was done, with or without short flaps. I like this present procedure. I think it is a wonderful one.

One feature that I like is that adductor muscles are utilized in closure. They are saved. The biggest trouble in the suction socket is loose adductor muscles.

I was happy to hear the talk about the absence of a tourniquet. I hate tourniquets in amputations. In vascular cases, you are more likely to do a rapid amputation without a tourniquet. I was glad to hear, in the technique, that there is no stripping of the periosteum. All it does is traumatize. There is going to be one-fourth inch of bone absorption. I mentioned the adductor muscles. One principle used in dressing is the use of traction and sensible compression, and there is no reason today why a good amputation stump cannot be fitted with a prosthesis within five to six weeks' time, provided there is no complication.

I congratulate the author on his presentation.

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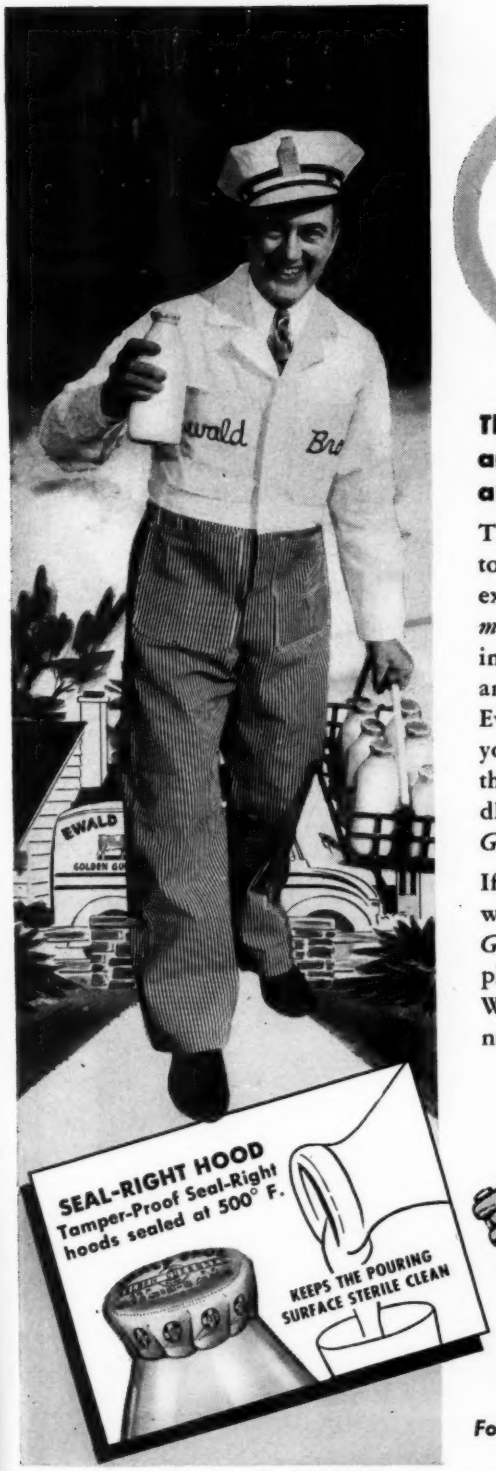

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G. H. Schroeder,
Business Manager

COLONIAL HALL—
One of the 14 Units in "Cottage Plan."